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MELANOSIS COLI

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Klerksdorp

Die verskynsel van melanosis coli skyn baie seldsaam te wees en na aanleiding van 'n geval wat onlangs onder my aandag gekom het, sal dit die moeite loon om die onderwerp in hersiening te neem en ook die geval te rapporteer.

Melanosis coli is 'n toestand wat ontstaan a.g.v. pigmentasie van die mucosa van die kolon deur melanien of 'n melanien-agtige pigment. Die eerste beskrywing is dié van Cruveilhier,[1] wat in 1829 by 'n lykskouing gevind het dat die mucosa van die kolon so swart soos Indiese ink was. In 1847 het Virchow[2] 'n geval beskrywe en eers in 1858 het hy die benaming van melanosis coli daaraan gegee.

Volgens beskrywings van Speare,[3] asook dié van Perkel en Troast,[4] varieer die kleur van die pigment vanaf grys na bruin en swart. Verskillende waarnemers het dit beskrywend vergelyk met dié van 'n paddavel, slang, krokodil of tiervel; 'n beskrywing wat, volgens diegene wat melanosis coli in sy verskillende stadia waargeneem het, heeltemal toepaslik is. Die pigmentasie mag eengalig of kollerig wees en kom vanaf die ileocaecalis-klep tot by die rektum voor. Proktoskopies bied dit dan ook 'n opvallende beeld.

Mikroskopies word die pigment in die sitoplasma van die groot mononuklêre selle in die tunica propria van die kolon gevind. Geen pigment word in die epiteelselle aangetref nie. Soms kom die pigment ekstrasellulêr voor.

Die etiologie van melanosis coli is nog nie met sekerheid vasgestel nie en uiteenlopende sienswyse kom in die letterkunde voor aangaande die ontstaan van die pigmentasie. Nietemin stem almal ooreen dat chroniese hardlywigheid die primêre oorsaak van melanosis coli is. Virchow was onder die indruk dat die kleurstof van bilirubien en hemosiderien afkomstig is. Pick[5] het in 1911 met die veronderstelling gekom dat die melanien afkomstig was van die reaksie van 'n ensiem op indol en skatol. In 1928 het Bartle[6] cascara sagrada genoem as die oorsaak van melanosis coli, en in 1933 het Bochus[7] getoon dat die antraseengroep van lakseermiddels, soos bv. cascara sagrada, senna, alwyn en rubarber, die oorsaak is. By 15 uit 18 gevalle het hy getoon dat die pigment binne 4 tot 12 maande verdwyn het nadat antraseen-lakseermiddels gestaak was. Verskillende waarnemers het daarna getoon dat cascara sagrada en verwante middels die oorsaak van melanosis coli was; Zobel en Susnow[8] het in 1935 7 gevalle gerapporteer. In 1940 het Roden[9] dit eksperimenteel met ape bewys en in 1941 het Bloch[10] melanosis coli by 3 uit 10 gevalle laat verdwyn deur onthouding van cascara vir 3 tot 6 maande.

Ten spyte van die bevindings, is dit nog nie onteenseglik bewys dat die antraseengroep-lakseermiddels wel die toestand veroorsaak nie, aangesien daar volgens Perkel en Troast, wel gevalle met melanosis coli voorgekom het wat selde enige van die lakseermiddels gebruik het, terwyl ander met chroniese hardlywigheid, wat antraseenmiddels gebruik het, nooit enige tekens van melanosis getoon het nie.

Dit is selfs beweer dat kunsmatige diarree, en nie soseer die pigment van die lakseermiddels nie, die oorsaak van melanosis coli is. Met ape, wat aan diarree, veroorsaak deur die toediening van cascara sagrada vir 5 weke, gely het, was dit moontlik om melanosis coli te veroorsaak, alhoewel dit nie met honde selfs na 1 jaar se inname van cascara sagrada gebeur het nie. Hier mag die argument aangevoer word dat die resultate aan die verskil van soort te wyte mag wees.

Na aanleiding van die veronderstelling dat die pigment van melanosis coli afkomstig is van voedsel ryk aan chlorofiel, het Hieronymi[11] uitgebreide eksperimente met plant-etende diere gedoen. Hy kon nie bewys dat die pigment wat in die mucosa van 3 gevalle van melanosis coli, sowel as by die betrokke diere teenwoordig was, aan chlorofiel te wyte was nie.

Speare haal 14 gevalle aan, waarvan 8 'n organiese vernouing gehad het en 6 'n sfinkterspasma as gevolg van vorige rektale skeure of hemorrhoïde operasies. Deur die toediening of onthouding van cascara sagrada, het Speare oor 'n tydperk van 15 jaar daarin geslaag om melanosis coli by 11 gevalle na willekeur te laat ontstaan of te laat verdwyn. Volgens die ondersoeke van Speare wil dit voorkom asof rektale obstruksie of ander obstruktiewe letsels van die kolon 'n baie vername rol by melanosis coli speel.

Speare het tot die gevolgtrekking gekom dat 'n fisiologiese gesonde mucosa geen pigment, wat melanosis coli veroorsaak, deurlaat nie. Die mucosa kan wel meer deurdringbaar word

as gevolg van obstruksie van die anus, ouderdom, herhaalde beserings deur toediening van klismas of gebruik van dilatore, en chroniese hardlywigheid. By dié gevalle veroorsaak die toediening van cascara bykans sonder uitsondering melanosis coli.

Op sigself skyn melanosis coli nie enige simptome te veroorsaak nie en klinies is dit in soverre belangrik, dat dit wel van melanotiese karsinoom onderskei moet word.

SKRYWER SE GEVAL

H.B., 'n blanke man van 74 jaar. Op 18 Januarie 1956 is hy met vermoedelike dermobstruksie tot die hospitaal toegelaat. Die geskiedenis wat hy gegee het, was dat hy vir baie jare, solank as wat hy kan onthou, aan hardlywigheid gely het. Hiervoor het hy altyd alwynpille gedrink wat hy soms met cascara sagrada afgewissel het. In Desember 1955 was hy ook behandel vir 'n verstopping wat verbeter het, maar nou het hy al bykans vir 'n week geen stoelgang gehad nie. Hy voel opgesit en is ook bewus van ongemak en pyn in die buik, veral aan die linkerkant. Die buikpyn het die dag voor opname begin en het stadig toegeneem. Hy was mislik, maar het nooit gebraak nie.

Met ondersoek is gevind dat hy van middelmatige bou was met swak spiertonus en tekens van gewigsverlies. Verder was sy algemene toestand bevredigend; hart en longe was normaal en die bloeddruk 160/100. Daar was 'n geringe mate van uitsetting, maar geen sigbare peristaltis nie. Met betasting was 'n volheid van die buik te bespeur en teerheid met ongemak in die linker hipochondrium. Dermklanke was duidelik waarneembaar en flatus is nou en dan gepasseer.

'n Flatus, sowel as 'n gewone klisma, was onbevredigend en het nie die uitsetting enigsins verminder nie. 'n Wangenstein, wat vir 3 dae opgestel was, het die uitsetting tot so 'n mate verminder, dat dit gestaak kon word en hy 'n lae-res-dieet kon neem. Na die toediening van agarol het hy ook 'n ontlasting gehad. Na 10 dae is 'n barium-klisma toegedien en die bevinding was as volg:

„Met die inlaat van die barium-mengsel het die dikderm normaal tot by miltboog gekanaliseer. Onder hoë druk het die barium uiteindelik drupsgewys deurgevloei nadat enorme uitsetting van die rektum, colon sigmoideus en descendens plaasgevind het. Die proksimale dikderm toon veelvuldige vullingsdefekte weens ontlastingsmateriaal.

„*Gevolgtrekkings:* Daar is bewys van gedeeltelike afsluiting van die colon transversus, proksimaal van die miltboog. Die aard van die letsel kan nie vasgestel word nie, maar is bes moontlik 'n lokale vernouing of bandverklewing."

Daar is toe besluit om 'n laparotomie te doen, met die oog op 'n reseksie. Op 6 Februarie 1956 is die buik deur 'n linker paramediale snit geopen. 'n Ringvernouing, net distaal van die miltboog, is gevind. Daar was geen vergrootte kliere of infiltrasie van die mesokolon bespeurbaar nie. Ongeveer 3 duim van die kolon is verwyder. Wat mens dadelik getref het, was die kleur van die mucosa: dit was egalig pikswart, proksimaal sowel as distaal van die vernouing. 'n Anastomose is gedoen, en die na-operatiewe verloop was sonder enige terugslag. Vyf dae na die operasie het hy 'n stoelgang gehad. Sy algemene toestand het algaande verbeter, maar hy het hardlywig gebly, sodat dit van tyd tot tyd nodig was om lakseermiddels toe te dien.

Hy is uit die hospitaal ontslaan met die versoek om binne 'n paar maande weer te rapporteer, maar daar is nooit weer van hom verneem nie, waarskynlik omdat hy op 'n afgeleë plek gewoon het en genadebrood geëet het.

Patalogiese Verslag. Die verwyderde kolon het 'n ringvernouing getoon wat nie die punt van die pinkie kon deurlaat nie. Makroskopies was die voorkoms dié van 'n karsinoom, maar die patalogiese verslag het geen melding gemaak van enige maligniteit nie, maar het gelui dat dit 'n geval van melanosis coli was.

OPSOMMING

'n Geval van melanosis coli word aangehaal, met verwysing na die bevindings van verskillende skrywers. Die gevolgtrekkings staaf ook die bevindings van andere, nl.:

1. Melanosis coli is 'n toestand wat a.g.v. pigmentasie van die mucosa van die kolon deur melanien of melanienagtige stof ontstaan. Die etiologie is nie heeltemal bekend nie, maar dit gaan altyd met chroniese hardlywigheid gepaard.
2. Cascara sagrada of een van die verwante antraseenlakseermiddels skyn die oorsaak van die pigment by melanosis coli te wees.
3. Op sigself veroorsaak melanosis coli geen simptome nie, maar kan met ander patologiese veranderinge gepaard gaan, soos bv. 'n vernouing, wat die pigmentasie vererger.

SUMMARY

A typical case of melanosis coli is reported in relation to the findings of various writers. This case bears out these findings. The conclusions arrived at are as follows:

1. Melanosis coli is a condition characterized by pigmentation of the mucosa of the colon by melanin or a melanin-like pigment. The etiology is not entirely clear, but chronic constipation appears to play a major part in the pathogenesis of melanosis coli.
2. Cascara sagrada or one of the related compounds of the anthracene group of laxatives appears to be the source of the pigment found in melanosis coli.
3. *Per se*, melanosis coli does not produce any symptoms, but is usually associated with some other pathological conditions, e.g. a stricture, which increases the intensity of the pigmentation.

VERWYSINGS

1. Cruveilhier. Aangehaal uit Speare[3] en uit Perkel en Troast.[4]
2. Virchow. Aangehaal uit Speare[3] en uit Perkel en Troast.[4]
3. Speare, G. S. (1951): *Melanosis coli. Experimental observations on its production and elimination in twenty-three cases.* Amer. J. Surg., **82,** 631.
4. Perkel, L. L. en Troast, L. (1951): Rev. Gastroent., **18,** 488.
5. Pick, L. (1911): Berl. klin. Wschr., **48,** 840 en 884.
6. Bartle, N. J. (1928): Med. J. Rec., **127,** 521.
7. Bochus, H. L., Willard, J. H. en Bank, J. (1933) J. Amer. Med. Assoc., **101,** 1.
8. Zobel, A. J. en Susnow, D. A. (1935): Arch. Surg., **30,** 974.
9. Roden, D. (1940): *Melanosis coli, a pathological study: its experimental production in monkeys.* Irish J. Med. Sci., p. 654.
10. Bloch, B. (1929): Amer. J. Med. Sci., **177,** 609.
11. Hieronymi, G. (1954): Zbl. allg. Path., **91,** 428.

ABSTRACT : UITTREKSEL

Lumbar Puncture Headache in relation to Age of Patient and Rest after Puncture. N. Rosedale. Brit, J. Vener. Dis., 1956, **32,** 127.

It was found in a group of over 400 punctures when a gauge-20 needle was introduced into the second or third interspace and 6 ml. of fluid were removed that those who rested for a few minutes on the abdomen and then walked a short distance to a ward and remained for 18 hours in bed the incidence of headache was 3·2% as compared with 17·3% among those who had to dress immediately and return to work after puncture. There did not appear to be any significant difference between the sexes in the incidence of headache. In patients under 40 years of age there was a particularly low incidence of headache among those rested.

F.W.F.P.

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EDITORIAL

TYPES OF CRETINS

It is only a few years ago that Jackson,[1] quoting the written authorities of the day, erroneously remarked in this *Journal* that 'cretins are of 3 types'. Stanbury and Querido[2] have summarized the present position with regard to the aetiological classification of cretinism. First they start by defining a cretin as 'a patient who has permanent retardation in development of the skeleton or central nervous system resulting from thyroid deficiency which existed during foetal or early neonatal life'. One criticism of this definition is that if a cretin is treated early enough the developmental retardation may be prevented from becoming permanent. The child was nevertheless 'a cretin' before treatment. Secondly it is noted that the authors omit consideration of juvenile myxoedema, in which hypothyroidism is acquired at an early age, frequently following an infective illness. The end-result of this state is indistinguishable from congenital cretinism, and although the words 'early neonatal life' in the authors' definition might seem to allow of its inclusion as a type of cretinism, yet they evidently exclude it.

Stanbury and Querido's first group is anatomic dysgenesis of the thyroid gland. This includes 'athyreosis', which is the usual cause of 'sporadic' cretinism in a non-goitrous area, and may produce 'congenital myxoedema'. Although usually looked upon as a non-inheritable disorder, yet multiple cases have been described in the same family. There is no thyroid tissue whatever, or merely a fibrous remnant containing an occasional thyroidal element. The degree of mental and physical retardation is severe. 'Congenital hypotrophic thyroid remnants' is a closely allied condition, in which there is believed to be an arrest of embryonic development of the thyroid gland. Radio-iodine localization methods have shown the thyroid remnants to be situated at the root of the tongue. These thyroid remnants are shown by iodine-131 studies to be hyperfunctioning.

'Cretinism from nutritional disorders' means iodine deficiency in an endemic goitre area. A large goitre is often present in the affected child, and there is always histological evidence of previous hyperplasia. The final clinical result is variable and depends upon the severity of intra-uterine damage, the functional capacity of the thyroid after birth, and the amount of iodine which becomes available to the

VAN DIE REDAKSIE

TIPES VAN KRETINS

Dit is slegs 'n paar jaar gelede dat Jackson,[1] toe hy uit die geskrewe werke van destydse gesaghebbendes aangehaal het, verkeerdelik in hierdie tydskrif verklaar het dat daar ‚3 tipes van kretins is'. Stanbury en Querido[2] het die huidige toestand, met betrekking tot die etiologiese klassifikasie van kretinisme, opgesom. Hulle het begin deur eerstens die kretin op te som as ‚'n pasiënt met permanente vertraging by ontwikkeling van die skelet of sentrale senuweestelsel as gevolg van skildkliergebrek, wat gedurende voorgeboortelike of vroeë nageboortelike bestaan voorgekom het'. Een rede waarom hierdie beskrywing gekritiseer word, is dat indien 'n kretin vroeg genoeg behandel word, dit verhoed mag word dat die ontwikkelingsvertraging permanent word. Die kind was nogtans ‚'n kretin' voor behandeling. Tweedens is dit opgemerk dat die skrywers nalaat om jeugmiksedeem, waarby skildkliergebrek op 'n vroeë ouderdom ontwikkel, dikwels volgende op 'n aansteeklike siekte, in ag te neem. Die eindresultaat van hierdie toestand kan nie van aangebore kretinisme onderskei word nie, en alhoewel dit mag voorkom of die woorde ‚vroeë nageboortelike bestaan' in die skrywers se definisie toelaat dat dit as 'n tipe van kretinisme ingesluit word, sluit hulle dit nogtans klaarblyklik uit.

Stanbury en Querido se eerste groep is anatomiese wanvorming van die skildklier. Dit sluit ‚atireose' in, wat die gewone oorsaak van ‚sporadiese' kretinisme is by 'n gebied waar skildkliervergroting weinig voorkom, en mag ‚aangebore miksedeem' veroorsaak. Alhoewel dit gewoonlik as 'n nieoorerflike siekte beskou word, is daar nogtans veelvuldige gevalle by dieselfde familie beskryf. Daar is hoegenaamd geen skildklierweefsel nie of slegs 'n veselagtige oorblyfsel met 'n toevallige skildklierelement. Die graad van verstandelike en liggaamlike vertraging is ernstig. ‚Aangebore hipotrofiese skildklieroorblyfsels' is baie na aan hierdie toestand verwant, en die mening is dat daar 'n stuiting van embrionale ontwikkeling van die skildklier by hierdie toestand is. Radiojodium-lokaliseringmetodes het getoon dat die skildklieroorblyfsels by die wortel van die tong geleë is. Studies met jodium-131 toon dat hierdie skildklieroorblyfsels oormatig funksioneer.

‚Kretinisme as gevolg van voedingsversteurings' beteken jodiumtekort by 'n gebied waar skildkliergeswel endemies voorkom. 'n Groot skildkliergeswel is dikwels by die aangetaste kind aanwesig, en daar is altyd histologiese bewys van vorige hiperplasie. Die finale kliniese resultaat is wisselbaar en hang af van die ernstigheid van besering in die baarmoeder, die funksionele kapasiteit van die skildklier na geboorte, en die hoeveelheid jodium wat aan die pasiënt beskikbaar is.

patient. Recent studies have shown that the thyroid glands of these cretins are able to accumulate iodine and produce thyroid hormone—even hyperthyroidism has been described.

'Inborn errors of metabolism' as a cause of cretinism includes several recently analysed conditions, involving absence or deficiency of one or other controlling enzyme. Since it is believed that enzymes control all the steps from the initial trapping of iodine in the gland, through the synthesis of thyroid hormone, to its final release into the blood stream, it can be seen that there is plenty of scope for nature's experiments and omissions. Patients in this group have in common enlarged hyperplastic thyroid glands from birth or early years of life. They are thus sporadic goitrous cretins. There are often several cases in one family.

In one metabolic variety, the iodine is trapped by the gland, and there is then a failure to combine it into organic compounds. In one case of this variety direct chemical assay of the iodine in the gland indicated that it was entirely in inorganic form. Before iodine can become incorporated with tyrosine it has to be oxidized to elemental iodine, probably under the influence of an oxidase. This latter enzyme is believed to be the missing link in these cases. In one family, 4 siblings were affected and the parents were first cousins—the inheritance involved therefore is possibly Mendelian recessive.

Another link in the chain leading to thyroid hormone is the coupling of mono-iodotyrosine and di-iodotyrosine in various combinations to form di-iodothyronine and tri-iodothyronine, of which there are several isomers. Thyroxin is formed from two molecules of di-iodotyrosine. In the case of two goitrous sisters it appears that this coupling of iodotyrosine was defective; thus ^{131}I uptake and retention was high, the ^{131}I could not be discharged from the gland with thiocyanate, and in the thyroid gland there were large amounts of the iodotyrosines but hardly any thyroxin.

The final type of metabolic disturbance is more complicated. In the normal course of mobilization of tri-iodothyronine and thyroxin from thyroglobulin, the latter undergoes proteolysis under the influence of a protease and the two active hormones are released into the plasma. Mono- and di-iodothyronine are also released from thyroglobulin and are normally de-iodinated under the influence of a specific de-iodinase which is present in the thyroid gland and other tissues. A lack of this enzyme would then allow escape of the iodotyrosines into the blood and actually a loss of these substances from the body. In this way a continued loss of hormone precursors could be brought about, with consequent insufficient bricks from which to build new thyroid hormones, and resultant cretinism with hyperplastic goitre. Several cases have been described in which this defect apparently occurred. In these patients ^{131}I rapidly accumulated in the glands but rapidly left them. Large amounts of di- and tri-iodotyrosines circulated in the blood. The patients' metabolism, and slices of the thyroid gland from one of them, were unable to de-iodinate extrinsic di-iodotyrosine. This hypothesis of the causation of cretinism in these several affected families seems highly probable, and

Onlangse studies het getoon dat die skildkliere van hierdie kretins in staat is om jodium op te gaar en skildklierhormoon voort te bring—selfs oormatige skildklierwerking is beskryf.

‚Aangebore foute van metabolisme' as 'n oorsaak van kretinisme, sluit verskeie onlangs-ontlede oorsake in, waarby afwesigheid van, of gebrek aan een of ander beherende ensiem betrokke is. Aangesien die mening bestaan dat ensieme al die stappe vanaf die aanvanklike vasvang van jodium in die klier, deur die sintese van skildklierhormoon tot by die uiteindelike vrystelling daarvan in die bloedstroom beheer, is dit voor die hand liggend dat daar baie speling vir die natuur se eksperimente en weglatings is. Pasiënte in hierdie groep het vergrootte hiperplastiese skildkliere vanaf geboorte of die vroeë lewensjare in gemeen. Hulle is derhalwe sporadiese skildklieragtige kretins. Dikwels is daar verskeie gevalle in een familie.

By een metaboliese soort word die jodium deur die klier vasgevang, en is daar dan 'n gebrek aan die verbinding daarvan tot organiese samestellings. By een geval wat tot hierdie soort behoort het, het direkte chemiese ontleding van die jodium in die klier getoon dat dit heeltemal van anorganiese vorm was. Voordat jodium tot tirosien vermeng kan word, moet dit tot elementjodium geoksideer word, waarskynlik onder die invloed van 'n oksidase. Die mening is dat hierdie laasgenoemde ensiem die ontbrekende skakel by hierdie gevalle is. By een familie was 4 kinders aangetas en die ouers was neef en niggie—die oorerwing hier was dus moontlik Mendelian-resessief.

Nog 'n skakel in die ketting wat tot skildklierhormoon lei, is die verbinding van mono-jodotirosien en di-jodotirosien in verskillende kombinasies om di-jodotironien en tri-jodotironien, waarvan daar verskeie isomere is, te vorm. Tiroksien word van twee molekule van di-jodotirosien gevorm. By die geval van twee skildklieragtige susters skyn dit of hierdie verbinding van jodotirosien gebrekkig was; dus was ^{131}I-opname en terughouding hoog, die ^{131}I kon nie saam met tiosianaat uit die klier uitgeskei word nie, en was daar in die skildklier groot hoeveelhede van die jodotirosiene maar skaars enige tiroksien.

Die finale tipe van metaboliese versteuring is ingewikkelder. By die normale gang van mobilisasie van tri-jodotironien en tiroksien van tiroglobulien, ondergaan laasgenoemde proteolise onder die invloed van 'n protease en die twee aktiewe hormone word in die plasma vrygestel. Mono- en di-jodotironien word ook van tiroglobulien vrygestel en die jodium daarin word normaalweg onder die invloed van 'n spesifieke ontjodiseerder, wat in die skildklier en ander weefsels aanwesig is, ontneem. Op hierdie wyse kan 'n aanhoudende verlies van hormoonvoorlopers teweeggebring word, met gevolglike onvoldoende stene om nuwe skildklierhormone te bou, en resulterende kretinisme met hiperplastiese skildkliergeswel. Verskeie gevalle is beskryf waar hierdie gebrek oënskynlik voorgekom het. By hierdie pasiënte het ^{131}I vinnig in die kliere opgegaar, maar gou weer daaruit gegaan. Groot hoeveelhede van di- en trio-jodotirosiene het in die bloed gesirkuleer. Die pasiënte se metabolisme en stukke van die skildklier van een van hulle was nie in staat om die jodium uit uitwendige di-jodotirosien te ontneem nie. Dit skyn of hierdie hipotese van die veroorsaking van kretinisme by hierdie verskeie aangetaste families, hoogs waarskynlik is, en Stanbury en

Stanbury and his associates have done fine work in elucidating these complex problems.

1. Jackson, W. P. U. (1952): S. Afr. Med. J., **26**, 605.
2. Stanbury, J. B. and Querido, A. (1956): J. Clin. Endocr., **16**, 1522.

sy kollegas het goeie werk ter opheldering van hierdie ingewikkelde probleme gelewer.

1. Jackson, W. P. U. (1952): S. Afr. T. Geneesk., **26**, 605.
2. Stanbury, J. B. en Querido, A. (1956): J. Clin. Endocr., **16**, 1522.

'MEDICINE IN SOUTH AFRICA—MEDISYNE IN SUID-AFRIKA (1957)'

This volume, which is published as a supplement to the *South African Medical Journal*, has been sponsored by the South African Council for the Exchange of Medical Sciences. It consists of a series of papers (in English) by South African authors, which have been published in the Portuguese Medical Journal *O Medico* under the auspices of the S.A. Council for the Exchange of Medical Sciences and under an Editorial Board consisting of Professors G. A. Elliott, J. Gillman, R. H. Goetz and S. F. Oosthuizen and Drs. J. Gear, E. Samuel and A. Shedrow.

The production reflects great credit both on the Editorial Board and on the printers. It consists of 157 pages of articles printed on art paper and contains a message from the Hon. J. H. Viljoen, Hon. President of the S.A. Council for the Exchange of Medical Sciences and Minister of Arts, Sciences, Education and Health, as well as a message from His Excellency The Portuguese Ambassador, General A. A. Pinto.

The following scientific articles appear:

Ebstein's anomaly of the tricuspid valve, by A. R. Bosman, Si Siew and G. A. Elliott.

Disorders of the parotid lymph glands simulating diseases of the parotid salivary glands, by D. J. du Plessis.

Epidemiological patterns of poliomyelitis in South Africa, by James Gear.

Widespread, non-calcifying lesions produced by Vitamin D with special reference to the role of the kidney in regulating the integrity of tissues, by J. Gillman and Christine Gilbert.

Studies on the haematological reactions produced in rats by prolonged treatment with trypan blue, by T. Gillman.

Experimental test with Battezzati-Tadei artificial kidney in the treatment of acute renal insufficiency, by A. Leonsins and *Piero Confortini*.

Pseudo-coarctation of the aorta, by Eric Samuel and L. Morris.

The use of operative and post-operative cholangiography in biliary surgery, by C. A. R. Schulenburg.

Duodenal ulcers associated with other conditions. Discussion on causation of associated syndromes. Outline of a new concept of gastro-duodenal ulcers, by A. Shedrow.

Dupuytren's contraction in diabetes mellitus, by T. Schneider.

Why atherosclerosis? by A. P. Walker and I. Bersohn.

Surgery of occluded Fallopian tubes, by W. Weinberg.

Raynaud's disease: A critical review of its diagnosis and treatment, by R. H. Goetz.

Pyelometry and further observations on hydronephrosis, by W. E. Underwood.

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FLUID AND ELECTROLYTE BALANCE PROBLEMS CONCERNING THE ANAESTHETIST*

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The anaesthetist's main concern with fluid balance is the pre-operative assessment of the patient's condition, and the administration of fluids, including blood, during surgery and in the early postoperative period.

Besides the usual clinical examination, evaluation of the water and electrolyte balance and of the nutritional status is essential in patients about to undergo surgery. Depletion of water, of sodium and of potassium, acid-base imbalance and hypoproteinaemia render the surgical patients vulnerable to operative trauma, and the correction of pre-existing deficits may become extremely difficult in the post-operative period on account of other complicating factors.

The patient who has been eating and drinking normally up to the time of admission is obviously in fluid balance and requires only replacement of the losses that occur during and after the operation. An average adult loses approximately 2,500-3,000 c.c. of fluid daily, of which about 1,000 c.c. is lost through the lungs and from the skin (insensible losses). Since the insensible losses occur through evaporation, the fluid lost is practically distilled water and contains only negligible amounts of electrolytes. During operation about 300 c.c. of water is lost per hour. If sweating is marked, the water loss may increase to 500-1,000 c.c. Sweat normally contains about 50 mEq. of sodium per litre.

If it is anticipated that oral feeding will be impossible for 2 days or more after surgery, it is advisable to start parenteral fluid administration during operation and so maintain the patient in fluid balance from the start. The quantity and composition of the fluid to be administered on the day of operation is determined by the losses that occur during this period. The metabolic response to trauma in the early post-operative phase is dominated by the increased secretion of suprarenal-cortical and pituitary-antidiuretic hormones. Therefore, on the day of the operation and on the first day following, there is retention of water, of sodium and of chloride associated with increased excretion of potassium. In spite of the sodium retention, plasma sodium is low. Hyponatraemia at this stage is physiological and explained by the diluting effect of retained water, a shift of sodium into the cells to replace lost potassium, and possibly by storage of sodium in the bones. Urinary output is reduced, (usually to 700-800 c.c.) and it is wrong to force the intake of fluid with the object of raising the volume of urine. The specific gravity of the urine is high—about 1·020-1·024—but sodium excretion is low.

Because of the metabolic changes which normally follow trauma, the solution administered on the day of operation should contain little or no sodium. The fear of sodium administration in this period is based on the unfavourable results which ensue when the solution used is almost exclusively 'normal' saline. This is certainly contra-indicated and either sugar (dextrose or invert sugar) in water or sugar with 0·2% sodium chloride should be given. The rationale for giving a small amount of salt is that some sodium and chloride are lost in the urine and in the sweat, and the plasma is hypotonic; salt-free solution may therefore intensify the hypotonicity and predispose to water intoxication. The quantity of the solution administered on the first day should, as a rule, be 2,000 c.c. Of this, 1,000 c.c. should be sugar with 0·2% salt and 1,000 c.c. should be 5% sugar in water. During the operation 300 c.c. per hour of either solution are given, and the remainder should be given at a uniform rate during the rest of the day.

Pre-operative imbalance may develop acutely, as in peritonitis, intestinal obstruction, burns; or gradually, as in patients suffering from a tumour in the gastro-intestinal tract, or any disease which interferes with normal intake of food and fluid for any length of time. The more acute the water and salt loss, the more severe are the symptoms. In chronic cases there is usually time for pre-operative rehydration and correction of imbalance. Even in acute cases it pays, whenever possible to postpone the operation for a few hours in order to achieve at least partial correction of the water, salt and acid-base imbalances.

The assessment of the magnitude and quality of the existing depletion is based mainly on clinical signs and symptoms. Laboratory examinations, apart from some simple tests, are only required in exceptional cases. The past history gives very valuable information. The duration of the illness during which the intake of food and fluid has been diminished should be carefully established; it is usually possible to obtain reasonably reliable information on the intake during this period. The urinary output before admission indicates the presence and degree of water shortage. Urinary volume, specific gravity and pH (a wide-range indicator paper is satisfactory) should be accurately recorded during the hours or days after admission. A low urinary volume of high specific gravity indicates water shortage. Urinary-chloride estimation, which can easily be carried out at the bedside, discloses the presence or absence of salt depletion. If the

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chloride content, expressed as sodium chloride, is more than 3 g. per litre, salt depletion can be excluded, except for salt-wasting conditions, which are rare. When vomiting is reported it is important to obtain information about the frequency and quantity. If the patient vomits whilst under observation, the pH should be ascertained with indicator paper. If the vomit is acid, hypochloraemic alkalosis should be suspected. One should not omit to enquire whether the patient had sweated, and whether it was necessary to change his pyjamas or even the bed linen because of this. When changing of pyjamas becomes necessary, at least an additional 500 c.c. of fluid per day has been lost; change of bed linen becomes necessary when the amount of sweat exceeds 1,000 c.c. It is important to note whether the patient has a high temperature. With each degree Fahrenheit above normal the daily loss of water increases by about 10%. Since it is the insensible loss of almost pure water that fever increases, febrile conditions are apt to produce hypertonic dehydration, with high-plasma sodium.

When the patient is examined, thirst, loss of skin turgor, sunken eye-balls, and the expression of the face are noted. The dryness of the skin is best acertained by feeling the axillae and groins for the presence of perspiration.

Dryness of the tongue may be misleading because of mouth breathing; it is more reliable to pass the finger between the gums and cheeks. The respiration should be observed, and particular attention should be paid to its depth; hyperpnoea may draw attention to acidosis; together with a history of starvation or loss of fluid in diarrhoeic stools, it makes the diagnosis of acidosis more than probable. If these simple clinical investigations do not seem to supply satisfactory information on the patient's fluid and electrolyte balance, then serum-sodium, serum-potassium, non-protein nitrogen, and carbon-dioxide combining power, should be estimated.

It is customary to distinguish between pure-water, pure-salt and mixed depletions. Clinically the deficiency is almost always mixed, with water depletion predominating. There are, however, conditions when water loss occurs with exceptionally little salt loss. Such conditions may develop whenever insensible losses are extremely heavy; for example, in high fever and in the presence of acidosis. On the other hand, excessive salt losses resulting in hypotonic dehydration develop when water and salt have been lost and water only has been replaced, for example in severe diarrhoea or vomiting, when the intake is water.

DIMINISHED PLASMA VOLUME

Generally speaking, the most significant disturbance resulting from water and salt depletion is reduction of the plasma volume and consequent circulatory and renal failure. The symptoms are more severe and appear earlier in salt deficiency and mixed depletion than when water only is lost. Salt loss is always accompanied by increased excretion of water and consequently is followed by water depletion which may be very severe. The blood pressure falls and the renal blood flow is reduced; the urinary output is small, and the specific gravity of the urine is high; blood urea or non-protein-nitrogen rises at an early stage when salt loss is severe. In salt depletion the kidneys conserve sodium and so the urine contains little or no sodium or chloride.

The dominating clinical symptom is that of circulatory impairment of various degrees, progressing to circulatory collapse. Examination of the veins of the hand gives useful information. Normally, the hand veins fill in the dependent position in 3-5 seconds and empty quickly in the elevated position. If after elevating the arm for a few seconds and lowering it below the level of the auricles the filling of the veins is slow or even absent, this is a sign of diminished plasma volume. This sign points to impending circulatory failure and is evident before the blood pressure drops.

The behaviour of the blood pressure can be used for the approximate estimation of the degree of dehydration and salt loss. If definite signs of dehydration are present and the systolic blood pressure is above 100 mm. Hg., one can assume that the loss is about 3% of the body weight. Giddiness, lassitude (often nausea) and a blood pressure of 90-95 mm. indicate fluid loss amounting to about 5-8% of the body weight. The blood pressure falls to below 90 mm., with postural fainting, apathy and vomiting when the fluid loss exceeds 10% of the body weight. The estimation of the magnitude of the fluid loss by these means is obviously a crude approximation only, but it is sufficiently accurate in most cases to allow fluid treatment to be started. In slowly developing, chronic dehydration the symptoms are less marked than in acute dehydration. The weight lost within a short time is also a useful guide, if the weight before the onset of the illness is known. One may assume that any weight loss which occurs within 1 week is caused mostly by water loss; however, in severe, especially febrile, diseases and after trauma, rapid oxidation of fat also contributes to the weight loss.

Acute dehydration of almost incredible magnitude develops rapidly in cases where translocation of large amounts of fluid takes place. Such cases are burns, peritonitis and intestinal obstruction. These patients are often deeply shocked. All signs of diminished plasma-volume and of circulatory failure may be present. It has been thought that the reduction of circulating blood volume was caused by a nervous reflex or by excessive production of histamine, which cause excessive dilatation of the vascular bed, so that the patient was, so to speak, bleeding into his own portal circulation. This may partly indeed be the cause of the shock, but it has definitely been observed that in peritonitis a considerable amount of fluid accumulates in the peritoneal cavity. In intestinal obstruction the fluid accumulates in the lumen of the intestinal loops above the obstruction. After severe mechanical or thermal injury, oedema-fluid accumulates in the injured tissues and their neighbourhood. In one published case of acute intestinal obstruction, for example, 500 c.c. of fluid was aspirated from the peritoneal cavity and over 3,500 c.c. from the intestine. In this case the measured fluid loss was 4,000 c.c., which in a person weighing 150 lb. would be as much as 6% of his body weight. The withdrawal of such an amount of fluid from the circulation within a few hours causes a reduction of circulating blood volume severe enough to explain the symptoms of oligaemic shock. Similar considerations apply to patients suffering from peritonitis or from severe trauma such as fracture of the femur and crush injuries. Restoration of the fluid balance should be carried out pre-operatively whenever possible; even partial replacement of the lost fluid improves the chances of recovery considerably. Patients in the stage of water, salt and potassium depletion whose glycogen

stores have been exhausted through starvation or in consequence of trauma are poor operative risks.

Treatment in chronically dehydrated patients is somewhat different from the measures to be taken in acute cases. The history of an imaginary patient suffering from stenosing pyloric ulcer is an example. He managed to keep in reasonably good health until, about a week before admission, vomiting set in. He lost large amounts of gastric juice and his intake was most inadequate. By the time of admission he had lost considerable amounts of fluids and clinically appeared seriously dehydrated. Besides water he had lost chloride in excess of sodium because his gastric juice was acid. Thirst had induced him to drink water and most of it was vomited. (Fluids are rendered isotonic before absorption from the gastro-intestinal tract; if water is taken salt will be secreted into the lumen and the fluid thus becomes isotonic with the plasma before absorption). In this patient with pyloric stenosis the onset of the vomiting which followed intake was delayed, and the salts secreted into the stomach were vomited and lost. He had thus lost large amounts of sodium, chloride and potassium, together with water. The chloride and potassium depletion had led to metabolic alkalosis.

TREATMENT

In such a patient the degree of dehydration, i.e., the amount of fluid lost, is estimated by the clinical signs and symptoms. Replacement is started with the administration of half-isotonic saline with dextrose. The physiological excess of chloride in sodium chloride helps to correct alkalosis but final correction cannot be achieved unless potassium is replaced.

As soon as the urinary volume is adequate, i.e. when it reaches 30-50 c.c. per hour, a solution containing sodium chloride in half-isotonic concentration (0·45%), potassium 0·3% and sugar 5-10% is administered. If the calculated deficit is more than 3-4 litres, replacement should be spread over 2 days. In chronically-ill, malnourished patients dehydration is almost always associated with potassium deficiency, anaemia and hypoproteinaemia. Whenever feasible, operation should be postponed for about 2 weeks, well spent on restoration of the water, electrolyte and nutritional balance. Treatment, which may be out of the hands of the anaesthetist, consists of the administration of water, sodium chloride, and potassium together with adequate calories and nitrogenous nutrients such as protein hydrolysate. Frequently-repeated small transfusions of about 250 c.c. of whole blood are of great help. In contrast to the beneficial effect of transfusions in these patients, preventive transfusion in persons with normal blood volume and cell count is of little value. Protein is metabolized or stored and the cellular elements are rapidly eliminated from the blood stream. Whether the solutions are given parenterally or by stomach tube, it is essential that protein hydrolysate should be preceded and combined with an ample supply of calories in the form of sugar. In the absence of adequate caloric intake amino nitrogen cannot be utilized; it is excreted, and only increases the urinary-solute load. If this point is overlooked, additional water loss and electrolyte imbalance may result. The increased urinary-solute load requires the excretion of large amounts of water and, urinary output being large, the false impression may be created that the patient is well hydrated, whereas in fact progressive dehydration is taking place. Furthermore, when the excretion of nitrogenous waste products is excessive, sodium is retained and this, together with the large water output, may give rise to hypertonic dehydration. The aim should be to supply about 2,000 calories per day in the form of sugar. If parenteral treatment is necessary, solutions containing 5% of alcohol in addition to 10% of invert sugar are at present the best for caloric supply.

If an operation has to be performed on an acutely dehydrated patient, for example for intestinal obstruction, peritonitis or extensive injury, when fluid loss is caused by the translocation of fluid masses, fluids should be administered as soon as possible before and also during the operation. In the assessment of the degree of fluid loss it should be borne in mind that in rapidly developing dehydration the symptoms are more severe than when the same amount of fluid is lost gradually.

In the presence of fluid translocation the fluid trapped in the 'third body space' must first be replaced with a solution whose composition approaches that of the plasma. Ringer-lactate solution is most suitable for this purpose. In addition to replacement of the existing loss with this solution, 1,500-2,000 c.c. of sugar in water should also be given in order to replace obligatory daily losses.

There are conditions of interest to the anaesthetist which deserve special consideration.

Potassium Depletion

In the average surgical patient, the administration of potassium is usually begun on the first or second post-operative day, when he is already out of the hands of the anaesthetist. Pre-operative replacement of potassium is necessary in surgical subjects with pre-existing potassium depletion. Potassium depletion, apart from its possible grave consequences during the post-operative course, is considered by several expert anaesthetists to be the cause of refractoriness to neostigmine in curarized patients. Detailed discussion of this condition is beyond the scope of this lecture; only a few salient points may be mentioned:

It has been known since the pioneer work on ionic milieu that potassium plays an important part in neuromuscular activity. In physiological experiments, reducing or increasing the concentration of potassium outside the physiological range results in blocking of the impulses. This shows an apparent analogy with the effect of potassium on the heart muscle. Both abnormally low and high potassium concentrations cause cardiac irregularities and finally arrest. If the potassium concentration is excessively high, the heart stops in diastole; if low, in systole. When autonomic ganglia or motor nerve fibres are stimulated, potassium is liberated into the perfusion fluid. Skeletal-muscle function is also closely associated with potassium, as is shown by the experience that intra-arterial injection of potassium salts causes powerful muscular contraction.

In familial periodic paralysis, an attack of paralysis develops when serum potassium falls below 3 mEq per litre. Administration of potassium salts achieves recovery, which begins about 10 minutes after the administration and reaches its maximum after approximately 7 hours.

Potassium antagonizes the action of curare. In experimental animals, after response to motor nerve stimulation had been abolished by curare, injection of potassium salts restores the muscle's reponse. This effect is transient and repetition of the injection of potassium salts again results in response to

stimulation. The sequence of curare action is that the effects of nerve stimulation and of acetylcholine are first abolished whilst injection of potassium salts will still elicit muscular contraction. At a later stage the effect of potassium is also abolished.

The reported cases of neostigmin-resistant curarization were mostly in debilitated, undernourished old patients. In these subjects potassium depletion is very frequent and in the majority, when serum-potassium estimations have been performed, low values were found (1·8-3·9 mEq. per litre). It should be remembered that the serum-potassium level is not always a reliable indicator of the intracellular potassium concentration. In one reported case electrocardiographic signs of potassium depletion were found. The published history of a certain aged woman is interesting: She had two episodes of intestinal obstruction caused by adhesions, within one month of each other. On the first occasion anaesthesia with the use of relaxants was uneventful but at the second operation curarization was resistant to neostigmin. Routine blood chemistry was performed on both occasions and it was found that serum potassium was 5 mEq. per litre at the first operation and 3 mEq. per litre at the second, when neostigmin resistance developed. Finally, the case in which neostigmin-resistant curare action was abolished by the rapid intravenous administration of 4 g. of potassium chloride seems to confirm the antagonism between the two agents which is known to exist in animal experiments.

Whether neostigmin resistance in curarized patients is due to prolonged myoneural blocking (for which there is some evidence), or to a central action of curare in the presence of potassium depletion through inefficiency of the blood-brain barrier, or to some unrelated mechanism, cannot be decided at present. If potassium depletion is accepted as a possible cause of neostigmin resistance, the logical conclusion is that, in patients in whom potassium deficiency is present, it should be corrected before the administration of curare. Should one decide to inject large doses of potassium chloride in a case of established neostigmin resistance, this should be done under frequent electrocardiographic control.

Shock

The outstanding features of shock are depression of all functions of the central nervous system, and circulatory failure apparently caused by a disparity between the circulating blood volume and the capacity of the vascular bed. In fluid therapy, restoration of the blood volume is the immediate aim of treatment.

If shock is caused by acute blood loss, replacement with whole blood is the logical treatment. The sooner blood transfusion is instituted after blood loss the greater is its efficacy. The rarely-attained ideal is to replace blood loss as it occurs. If transfusion is started simultaneously with the blood loss, volume-for-volume replacement will prevent shock; if 2-3 hours have elapsed between the blood loss and the beginning of transfusion, 2-3 times the volume lost must be given in order to maintain blood pressure. If transfusion is initiated 7-8 hours after the accident leading to blood loss, repeated large transfusions may not be sufficient to restore and maintain blood pressure. Loss of blood is not the only cause of haemorrhagic shock. Loss of blood amounting to 25% of the total blood volume may not by itself cause permanent fall of the blood pressure, but when combined with trauma it may easily induce shock.

After blood transfusion, it is usually necessary to administer fluid to maintain blood pressure. Sugar solution without salt is ineffective. A solution containing 0·45% salt with sugar should be given. Administration of sugar, preferably invert sugar, is necessary because in shock, after initial hyperglycaemia, blood sugar soon falls to hypoglycaemic level and continues to fall as shock progresses to irreversibility.

In shock caused by dehydration of whatever origin (diarrhoea and/or vomiting, together with urinary and insensible losses), an initial infusion of Ringer-lactate solution should be given, followed by multiple electrolyte solution, hypotonic with respect to sodium chloride. Plasma or whole blood should also be given if shock is severe or hypoproteinaemia is present. It is recommended that blood should be given after the initial administration of 1,000 c.c. of Ringer-lactate solution.

In accordance with the view that in shock there is a disparity between the circulating blood volume and the capacity of the vascular bed, vasopressor drugs are added to the intravenously administered solution. The usual dose is 8 c.c. of a solution of 1 : 1,000 adrenaline hydrochloride or noradrenaline to 1,000 c.c. of the injection-solution. The rate of administration is determined by the blood-pressure response.

Burns

The outstanding change in fluid metabolism after burn injury is the rapid translocation of fluid into the burnt area. The amount of fluid lost is proportional to the extent of the burn. It can therefore be calculated, and it is important that the full amount required should be given but no more. The extent of the burnt area is calculated by the 'rule of nines,' according to which the body is divided into parts each representing 9% or a multiple thereof of the total body surface. Head 9%, one upper limb 9%, front of the trunk 18%, back of the trunk 18%, each lower limb 18%, perineum 1%. It is a common error to overestimate the extent of the burnt area; and then the amount of fluid calculated overloads the circulation. As the fluid trapped in the injured area is similar in composition to plasma, the fluid of choice for replacement is plasma itself, if available; if not, Ringer-lactate solution should be used. Capillary damage causes shift of plasma protein into the interstitial spaces and proteinuria of varying degrees is also common. To correct the ensuing hypoproteinaemia a colloid should be given, preferably in the form of plasma or, if this is not available, dextran. Thermal injury destroys blood cells and the deeper the burn the more cells are damaged or destroyed. Consequently in deep burns whole blood must be transfused. Anaemia, often of a serious degree, regularly develops after burn injury; it was observed that anaemia is much less severe and is more easily corrected if whole blood is given during the early stages of treatment.

Solutions to be administered during the early treatment of burn are Ringer-lactate solution, colloids in the form of plasma or dextran, and whole blood. In addition, sugar in water (dextrose 5% or invert sugar 10%) must be given to cover continuous insensible losses. The amount of fluids required during the first 24 hours is calculated by the following formulae:

$$\text{Ringer-lactate solution (c.c.)} = \frac{\text{body weight (lb.)} \times \% \text{ burnt area}}{4}$$

$$\text{Colloid (c.c.)} = \text{body weight (lb.)} \times \% \text{ burnt area} \times 0{\cdot}75 \text{ c.c}$$

The volume of sugar in water to be given varies with the required amount of Ringer-lactate solution. If this is not more than 3,000 c.c., the volume of sugar in water should be 2,000 c.c.; if 3,500-4,500 c.c., then 1,500 c.c.; and if 5,000 c.c. or more, then 1,000 c.c.

In second-degree burns involving less than 20% of the body surface, colloid is not required; in deep burns if the extent is more than 20%, all the colloid used should be in the form of whole blood. It is most essential not to allow the burnt patient to lapse into deep shock, and therefore one must aim at replacing the loss of fluid at the rate that it occurs. The development of burn-oedema is rapid at first and in 8 hours about two-thirds of the total fluid that will form the oedema has been withdrawn from the circulation. Accordingly, one-half to two-thirds of the amount of fluids calculated for the first 24 hours (Ringer-lactate solution and/or colloid) must be given within 8 hours after the accident. The rest should be evenly distributed over the remaining hours of the first day. On the second day about one-half to two-thirds of the fluids administered on the first day are given, according to the condition of the patient. The administration of large amounts of fluid to burnt patients on the first day is in seeming contrast to the general rule that surgical or traumatized patients should receive only moderate volumes of fluid on the first one or two days. In burnt patients the translocated fluid must be replaced at the rate it is lost to the circulation. Also, whereas in the average traumatized patient oliguria is a normal response to trauma and a favourable sign, in burnt patients the hourly urinary output must be 30-50 c.c. Urinary output must be carefully watched and charted from the beginning and it is often necessary to introduce an indwelling catheter during the first 48 hours. Inadequate urinary output is a sign of insufficient administration of fluid, and if not corrected the patient will invariably pass into irreversible shock. Although the anaesthetist may not see the burnt patient after the first day, it is important to realize that when reabsorption of the translocated fluid begins, usually on the third day, intake must be restricted drastically or stopped altogether. The only indication that this is occurring is the increasing volume of urine. The intake of fluid then must be so adjusted that the hourly urinary volume remains between 30 and 60 c.c.

Head Injuries and Brain Surgery

Most neurosurgical patients require parenteral administration of fluid during surgery and afterwards. Increase of the cerebrospinal fluid pressure is often a serious complication and the margin between tolerated and fatal upper limits is narrow. It has been established that the quality of the administered fluid influences CSF pressure. Infusion of 5% dextrose in water invariably results in increased CSF pressure. The administration of 5% dextrose in 0·9% sodium-chloride solution reduces the CSF pressure, which, according to the rate of infusion, may fall to as low a level as 50-30% of the starting pressure. If 2·5% dextrose in 0·45% (half isotonic) saline is infused at an average rate no change in CSF pressure occurs. Infusion of 5% dextrose in water to patients with increased CSF pressure is particularly dangerous.

Disturbances of the sodium metabolism are not infrequent in diseases of the central nervous system or following injuries to the brain. The production of the pituitary antidiuretic hormone is under the nervous control of the paraventricular and supraoptic nuclei. If these, the connecting tract, or the posterior lobe of the pituitary, are damaged, a condition akin to diabetes insipidus may develop. Large amounts of hypotonic urine are then excreted with resulting hypernatraemia.

Loss of water and increased concentration of sodium and chloride in the blood may also occur through an entirely different mechanism. The stimulus of thirst is absent in the unconscious or semicomatose patient, who is unable to drink even if water is offered to him. The insensible loss of water which continues is greatly increased if fever or hyperpnoea is present.

Old Age

With the increasing frequency of surgery performed on old patients certain metabolic peculiarities of old age assume interest to the anaesthetist. In old age intracellular water content diminishes and the volume of extracellular water increases. Therefore extracellular dehydration cannot be promptly compensated at the expense of intracellular water. Salt depletion may occur in elderly persons without obvious loss of sodium. This is often caused by insidious, chronic malnutrition. The lowered serum-sodium concentration may produce no symptoms but, together with associated water loss, renders the aged patient markedly vulnerable to subsequent loss of electrolytes and water. Hyponatraemia may explain asthenia, mental confusion, azotaemia and hypotension in older patients. Hyponatraemia is frequently accompanied by potassium depletion. Obese old persons are particularly vulnerable to electrolyte imbalance. Water intoxication is a greater danger in older people than in young subjects. Accordingly, unless hypertonic dehydration exists, hypotonic salt solution with sugar is preferable to sugar in water.

On the other hand, aged persons are liable to develop hypertonic dehydration because of the reduced sensation of thirst, especially if consciousness is clouded. The concentrating power of the kidneys is diminished in old age and the urine more dilute. If replacement of water is inadequate, hypertonic dehydration develops. The excretory and reabsorptive capacity of the kidney tubules suffers with advancing age, and so the efficacy of renal control of hydrogen-ion concentration becomes impaired. Because of reduced pulmonary ventilation and increased residual air in the alveoli, carbon-dioxide elimination is reduced and the aged patient lives in a state of compensated respiratory acidosis. On account of the reduced efficacy of both renal and pulmonary control, the aged patient's acid-base balance is indeed labile.

Evaporation of water from the skin is diminished as compared to younger subjects and on account of reduced pulmonary ventilation loss of water through the lungs is also less. Accordingly the daily maintenance requirement of water is less—about 1,500-2,000 c.c. as compared with 2,500-3,000 c.c. for younger patients.

Frequently occurring undernutrition and hypoproteinaemia may require pre-operative preparation with large doses of sugar and protein hydrolysate. If anaemia is also present repeated small transfusions of whole blood before surgery are often desirable.

Shock easily becomes irreversible in old age and pre-operative preparation with correct early post-operative fluid

therapy can do a great deal to prevent it. Since compensatory mechanisms in the old organism are inefficient and the range of tolerable deviations narrow, serum chemistry before and after surgery is more often required in order to detect disturbances of the sodium and potassium metabolism and abnormalities in acid-base balance.

'STILLBIRTH'—A THREE-YEAR ANALYSIS OF THE CASES AT THE HOSPITALS IN THE OBSTETRICAL UNITS OF THE UNIVERSITY OF CAPE TOWN

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The definition of a stillbirth generally recognized in the United Kingdom is any child issued forth from its mother after the 28th week of pregnancy which did not at any time after being expelled breathe or show any other signs of life. The mere presence of the heart beat is regarded as a sign of live birth. In the obstetrical units of the University of Cape Town nothing less than pulmonary ventilation is regarded as a sign of live birth.

The following brief report is presented in an attempt, not only to analyse and classify as accurately as possible the causes of stillbirth, but also to explain the reasons for the high stillbirth rate at the above units for the 3-year period 1952-54, inclusive. It will be obvious that, because of the variation in definition, accurate comparisons in stillbirths with other centres is not possible and that the rates will be higher in the University units at the expense of the neonatal deaths. This, however, is but a minor reason for the higher loss at birth.

The obstetrical units of the University of Cape Town comprise 5 maternity hospitals, viz. the Peninsula Maternity Hospital providing for both White and non-White patients (Coloured and Bantu), Somerset Hospital, exclusively for non-Whites (Bantu and Coloured), St. Monica's Hospital, exclusively for non-Whites (Coloured and Bantu), Mowbray Maternity Hospital, exclusively for Whites, and Groote Schuur Hospital, almost exclusively for non-Whites but with a few White patients. The Groote Schuur Hospital maternity unit deals mainly with maternal diseases in pregnancy, viz. the toxaemias, all cases of eclampsia from the Univerity units, diabetics, etc., i.e. patients with the medical complications of pregnancy. The majority of the patients admitted to the Groote Schuur Maternity Unit were non-booked.

The accommodation of these hospitals was stretched to its limits by patients seen at regular antenatal clinics of the University units and by 'emergency' cases. In fact at no time was over-crowding not in evidence at the above institutions and very often patients in need of hospital care were turned away.

District patients were delivered by trained midwives attached to the above hospitals, which also catered for women seen in outlying antenatal clinics as far afield as 100 or more miles from the city.

MATERIAL

During the period of 3 years 19,702 women were delivered of 20,191 infants at the above hospitals. At the same time 23,411 children were born in both hospital and district. Of 900 stillbirths which occurred in hospital, 97 occurred amongst the 4,285 born to White women (a stillbirth rate of 22·6 per thousand), and 803 amongst the 15,906 born to non-White women (a stillbirth rate of 50·5 per thousand). The stillbirth rate for all races in hospital practice was therefore 44·5 per thousand and in hospital and district combined 38·4 per thousand (uncorrected). During the same period of time in private maternity institutions in Cape Town, 92 stillborn babies were delivered out of a total of 7,266 White infants, i.e. a stillbirth rate of 12·5 per thousand.

There were 16,819 infants delivered in 'booked' patients and 3,372 in 'emergency' cases, the 'emergency rate' being about 20% of all admissions. It should be stressed, however, that this latter figure failed to include many women who defaulted from attendance at the hospital antenatal clinics for as long as 2 or even 3 months after being 'booked in' at their first visit and who were not seen again until they were admitted with a serious complication of pregnancy or labour. It was my impression that the correct 'emergency rate' of admission to hospital was nearer 25% than 20%. Of the 'booked' patients, 405 infants were stillborn (24·6 per thousand), in which no foetal heart sounds were heard in 42·9% of cases. In the 'emergency' group, 495 babies were stillborn (146·8 per thousand), in which no foetal heart-sounds were heard on admission in 49·3% of cases. The loss in 'emergency' cases was therefore almost exactly 6 times higher than in 'booked' patients.

The seriousness of 'emergency' admissions was emphasized in the cases quoted by Lawson and Lister[2] from the Ibadan University College and in the perinatal mortality recorded by Thompson,[3] where in both instances the mortality was 5 times higher in 'emergency' than 'booked' cases.

CAUSES OF STILLBIRTHS

The causes of foetal death in the 900 stillbirths under consideration are classified in the following table:

A. MATERNAL CONDITIONS DURING PREGNANCY			
1. Antepartum Haemorrhage *			232 (25·8%)
(*a*) Accidental haemorrhage	178 (19·7%)		
With toxaemia of pregnancy or essential hypertension	119 (66·8%)		
Without toxaemia or essential hypertension ..	59 (33·2%)		
(*b*) Placenta praevia (with toxaemia of pregnancy in 1)	45 (5·0%)		
(*c*) Unexplained haemorrhage (circumvallate placenta in 2)	9 (1·0%)		
2. Toxaemia of pregnancy and essential hypertension		61	(6·8%)
3. Eclampsia (forceps in 12)		30	(3·3%)
4. Syphilis (maceration in 25)		26	(2·9%)
5. Diabetes and prediabetes (maceration in 6) ..		16	(1·8%)
Total A		365	(40·6%)

* Out of 232 cases of antepartum haemorrhage, 147 were premature births, of which 119 followed accidental haemorrhage and 28 placenta praevia.

B. HAZARDS OF LABOUR

1.	Umbilical cord complications		121 (13·4%)
	(*a*) Prolapse of cord	90 (10·0%)	
	(*b*) Cord round neck	21 (2·3%)	
	(*c*) Cord presentation	7 (0·8%)	
	(*d*) Knot of cord	2 (0·2%)	
	(*e*) Stenosis of cord (macerated)	1 (0·1%)	
2.	Primary breech delivery and extraction		57 (6·3%)
3.	Normal delivery, and after prolonged labour		42 (4·7%)
4.	Precipitate labour		5 (0·6%)
5.	Forceps extraction (not including cases of eclampsia)		41 (4·6%)
6.	Transverse lies (vaginal delivery)		34 (3·8%)
7.	Ruptured uterus (12 emergencies)		16 (1·8%)
8.	Caesarean section		12 (1·3%)
9.	Destructive operations (not including abnormal foetuses)		10 (1·1%)
10.	Impacted shoulders (vertex presentation)		4 (0·4%)
11.	Compound presentations		2 (0·2%)
	Total B		344 (38·2%)

C. UNKNOWN CAUSES

1.	Macerated stillbirths		101 (11·2%)
2.	Fresh stillbirths		41 (4·6%)
	Total C		142 (15·8%)

D. FOETAL MALFORMATIONS

1.	Hydrocephalus		12 (1·3%)
2.	Anencephalus		7 (0·8%)
3.	Other causes		7 (0·8%)
	Total D		26 (2·9%)

E. MISCELLANEOUS CAUSES

1.	Rhesus incompatibility (maceration in 6)		18 (2·0%)
2.	Extra-uterine pregnancy (macerated)		2 (0·2%)
3.	Short umbilical cord		1 (0·1%)
4.	Angioma placentae		1 (0·1%)
5.	Ruptured spleen		1 (0·1%)
	Total E		23 (2·5%)

Postmaturity as a cause of intra-uterine death and stillbirth is not included in this series of cases because of the unreliability of menstrual histories given by the majority of group-3 patients (see below). Baird, Walker and Thomson[5] found that the proportion of stillbirths rises from 60% before term to about 75% in cases 2 weeks or more postmature.

DISCUSSION

It was realized when tabulating the causes of stillbirth that the above clinical classification was not completely adequate because of non-inclusion of pathological reports, although many autopsies were performed. An ideal classification (to suit both obstetrician and pathologist) is still to be formulated, because of the considerable overlapping of factors in the causation of stillbirth. This difficulty was further emphasized by Morrison,[4] who found that, despite autopsy, about 40% of stillbirths revealed no cause of death, and very few pathologists will disagree with this statement. Baird, Walker and Thomson[5] found more meaning in a classification based on clinical findings than in a report based purely on pathological causation. They maintain that in the presence of a well-defined abnormality of pregnancy or labour it is reasonable to consider that this may be the cause of death, at least if the abnormality is sufficiently severe in the absence of any obvious inconsistency in such reasoning. In the obstetric units of the University of Cape Town asphyxia at post mortem was found to account for the majority of deaths in accidental haemorrhage, as was similarly reported by Drillien.[6]

The main factors, therefore, involved in stillbirth causation in the above series were seen to be divided into 4 main groups:

A. MATERNAL ILL-HEALTH

Maternal ill-health was concerned with the causation of 40·6% of stillbirths. Figured prominently in this group is prematurity, which most authors[5-10] agree is the greatest contributory factor in stillbirth loss. The significance of prematurity is well enough known to warrant further discussion in a paper of this kind. As internationally accepted, the definition of prematurity is any infant weighing at birth 5½ lbs. (or 2,500 g.) or under, without regard to the period of gestation. Macgregor[5] reported that 54% of stillbirths occurred in premature babies. In our units no fewer than 376 of the stillbirths were premature babies, i.e. 41·8%. Of these, 86 (9·6%) were 3 lb. or under. The underlying factors in the etiology of prematurity can be classified under 3 headings:

1. *Maternal Ill-health* such as toxaemia of pregnancy, including eclampsia, antepartum haemorrhage, essential hypertension, diabetes, syphilis and other acute and chronic diseases. In the above series 268 (29·8%) of stillborn infants were delivered from mothers afflicted with toxaemia of pregnancy and essential hypertension. Drillien[6] found a prematurity rate of 14·9% in an incidence of 14% toxaemia in her series.

2. *Multiple Pregnancy*. Record, Gibson and McKeown[11] found the stillbirth rate to be inversely related to the number of foetuses in the uterus. Many other authors[12-15] have recorded high stillbirth losses in twins born in hospital. The reasons for the larger wastage were noted to be (*a*) a higher prematurity rate; (*b*) a more frequent rate of toxaemia; (*c*) more complications of labour, such as prolapse of the cord, abnormal lies and the like; and (*d*) other causes, such as illegitimate births and parity.

(*a*) The University units report the abnormally high incidence of twins of 1 in 52·9 (373 sets of twins), in which there was a 55·5% rate of *premature babies* and a stillbirth rate of 7·4%. In the same period 8 sets of triplets (1 in 2,463) were born. Record *et al.*[11] noted that prematurity and atelectasis accounted for 64·4% of the loss in twin pregnancy, whereas in single pregnancy the wastage due to this factor was 10·5%.

(*b*) The *toxaemia* rate in twin pregnancy is higher than in single pregnancy and in our units 20·4% of the twin pregnancies were associated with toxaemia. Record *et al.*[11] similarly found a higher toxaemia rate in twins of 19·5% and in single pregnancy only 3·6%.

(*c*) The *complications of delivery* in multiple pregnancy are increased because of the increased frequency of prolapse of the cord, abnormal lies, uterine inertia and the like. Record *et al.*[11] reported a 39% stillbirth loss from this factor as compared with only a 11% wastage in single pregnancies.

Drillien[6] noted that during labour the wastage following mature and premature deliveries was as follows:

	Premature	*Mature*
Vertex	33·8%	1·6%
Vertex (all forceps)	33·3%	3·8%
Version and extraction	25·0%	4·4%
Breech	52·8%	15·1%
Caesarean section	36·7%	6·0%

(*d*) *Illegitimate births*, according to most authors,[1-15] are associated with a higher proportion of premature babies being born. In the City of Cape Town (1952-54)[16] the incidence of illegitimate births was found to be 3·4% in Whites and 27·5% in non-Whites. The stillbirth rate for legitimate European births was 16·3 per thousand and the corresponding rate in non-Whites 36·1 per thousand. Contrary to other reports, the stillbirth rate for illegitimate births in Whites and non-Whites in Cape Town were practically the same at 39·6 and 40·6 per thousand respectively.

Parity. Most teaching hospitals report a higher stillbirth rate where more primigravida are delivered and also when the parity extends beyond 3 or 4 confinements. This was found also at the teaching units of the University of Cape Town.

B. HAZARDS OF LABOUR

This, the second biggest killing factor in our obstetric units, accounted for 38·2% of the stillbirths. Sutherland[10] found the stillbirth wastage due to accidental haemorrhage and difficulties to be 33% in Scotland in 1943. When it is realized that the stillbirth wastage was practically 6 times higher in 'emergency' than in 'booked' cases in our obstetric hospitals, one can easily explain the high stillbirth loss resulting from the hazards of labour. Lawson and Lister[2] found a 5 times heavier stillbirth loss, and Thompson[3] a similar loss in perinatal mortality. Umbilical-cord complications head this group, accounting for as many as 121 stillbirths (13·4%), of which 90 were the result of prolapse of the cord. Of the cases of this, the only avoidable catastrophe, 53·7% occurred in emergency admissions, in 1/3rd of which foetal heart sounds were not heard. In only 68 cases (43%) did the infant survive out of a total number of 158 instances of prolapse during the 3 years. Cope[17] recorded a survival rate of 72·1% in an incidence of 0·58% at Leeds for the years 1938-47, 59% of these patients being unbooked. The large stillbirth loss due to cord prolapse could certainly be reduced by more frequent use of Caesarean section when cervical dilatation is incomplete, by routine vaginal examination at the onset of labour, and by more careful antenatal care and supervision of patients with conditions predisposing to prolapse of the cord.

STILLBIRTHS FROM OTHER CAUSES

C. *Unknown Causes*

There were 142 stillborn babies (15·8%) in this group. Of these, 101 (71%) were macerated, the cause of foetal death not being discovered, even on post-mortem examination, in the majority of cases. Is it possible that latent syphilis in a 'Wassermann-negative' woman, possibly treated previously for syphilis in an earlier pregnancy, might account for the large number of macerated infants? It is felt that many previously 'Wassermann-positive' patients who have had treatment in their first pregnancy are not advised to have repeated treatment in every subsequent gestation, as used to be recommended as a routine in the 'pre-penicillin' days. Of course one must not forget the large number of pregnant women who suffer from general ill-health apart from syphilis, particularly those living under conditions of extreme poverty, who might 'miscarry' or produce macerated infants.

D. *Foetal Malformations*

As compared with other centres there were very few malformed stillbirths in the 3 years 1952-54. Only 26 abnormal stillborn infants were born (2·9%), as compared with the figures of Macgregor,[8] who reported 20%, Drillien[6] 19%, and Baird, Walker and Thomson[5] 15·6%. No explanation can be offered for the marked difference.

E. *Rhesus Incompatibility*

In this group, 18 cases occurred (2%), in which 6 of the foetuses were macerated. The low incidence of Rh incompatibility in the Bantu as compared with the European was noted.

The comparative rarity of syphilis in causing stillbirths was another feature during the years 1952-54; only 26 stillborn babies (2·9%), of which 25 were macerated, were noted in the present series. It speaks well for our antenatal services that the majority of pregnant syphilitic women attending at the above hospitals are being treated as a routine measure.

CONCLUSIONS

During the past 15 years an obvious and striking fall in the stillbirth rates has occurred in the City of Cape Town, as can

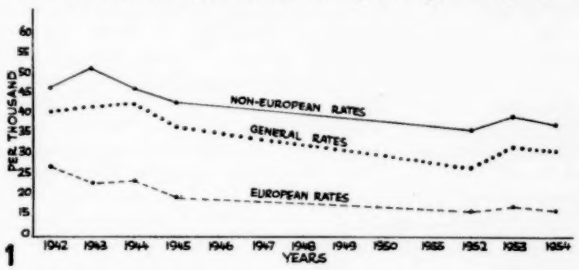


Fig. 1

be seen in Fig. 1, although the rates are still considerably higher than in oversea European and American centres. Two explanations for the higher stillbirth rates are offered:

1. More stillbirths are recorded at these units because of the difference in the definition of stillbirth (see above).

2. Despite the marked fall in the White stillbirth rate, there has been no such similar diminution in the non-White. This will be noted in Figs. 2A and 2B.

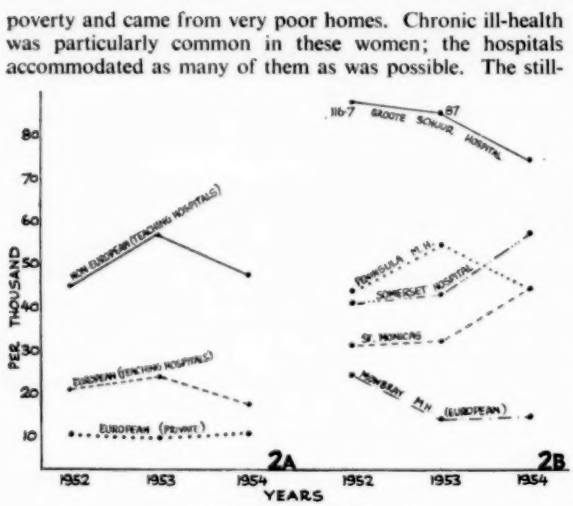
One may therefore classify the patients in this series into 3 main groups, depending on their socio-economic standing:

Group 1. Private patients (White), belonging to the upper socio-economic class and delivered in private institutions by specialists and private practitioners. The stillbirth rate in this group was only 12·5 per thousand for 3 years.

Group 2. The lower income group (or so-called middle class), consisting of both Whites and non-Whites. The stillbirth rate was 22·6 per thousand for 1952-54.

Group 3. The lowest income group, practically all non-Whites, the majority of whom lived in conditions of extreme

poverty and came from very poor homes. Chronic ill-health was particularly common in these women; the hospitals accommodated as many of them as was possible. The still-



Figs. 2A and 2B

birth loss in this group over the 3 years 1952-54 was 50·5 per thousand.

REASONS FOR THE HIGH STILLBIRTH RATE IN GROUP-3 PATIENTS

(*a*) *Socio-economic*

Almost all authors [18-21] are agreed that under conditions of poverty, poor nutrition, inadequate rest and poor hygiene, the stillbirth losses are much higher than in conditions of plenty. A superior diet will reduce the premature rate[18-21] with a consequently higher proportion of mature infants; the toxaemia of pregnancy incidence will fall, with a lessening of the number of cases of accidental haemorrhage, and general ill-health in the mother will naturally be markedly reduced. The strikingly marked improvement in infant losses in Scotland[10] during the last world war as a result of dietary improvement is ample evidence of what can be achieved.

Steer and Kasar[20] at the Sloane Hospital for Women reported the incidence of toxaemia in private patients (socio-economically 'well-off') to be 5 times less than in ward patients—patients consisting mainly of Europeans, Negroes and Puerto Ricans. The difference was not due to the Negroes but more to the Puerto Ricans, who were probably the poorest economically and hygienically and who exerted a real difference on the ward rates. Douglas[21] similarly stresses the socio-economic factors as a cause of a high prematurity rate and stillbirth loss.

The maternity units of the University of Cape Town show a similar state of affairs, because the bulk of their patients belong to the group-3 type. There is little hope that any substantial lowering of the present rates of obstetric mortality due to maternal ill-health can be accomplished unless vastly greater improvement in the socio-economic field can be attained or unless more intensive application of existing clinical knowledge can be forthcoming, expecially in the field of the causation of toxaemia.[5]

(*b*) *Intrapartum Care*

Baird *et al.*[5] maintain that the application of a very high standard in midwifery to a community which enjoys adequate and satisfactory antenatal care, helps to foster and maintain a low obstetric death rate, especially by reducing the death rate due to trauma or difficulties in labour. The stillbirth rates will consequently be reduced.

Despite 'apparent' good antenatal care at the units of the University, no such a reflection in the stillbirth figures occurred except in the Whites. The large numbers of 'defaulters' at the antenatal clinics of the above hospitals is still a factor in the admission of a fair number of 'complicated cases' which were not seen for as long as 2 or even 3 months after the time of the original first booking. These patients, together with the large numbers of non-booked women admitted to the University units, swelled the 'emergency' rate to nearer 25% than 20% of all patients admitted. The dangers of such types of patients, particularly in poor states of health, are reflected in the stillbirth rates, which are 6 times higher than in booked patients.

It must also be stressed that the extreme shortage of bed accommodation for the group-3 type of woman in our units has in no small manner helped to increase the above dangers. It has long been acknowledged that there are many women in need of institutional care who cannot obtain such supervision because of the overcrowded conditions at the maternity hospitals. It is maintained that a large number of the complicated cases seen at the hospitals concerned in this series might have been prevented had more beds been made available for the large maternity population (particularly group-3 women) of Cape Town. One realizes, too, that the greatest hindrances to good antenatal care are the large numbers of women that must be seen at the average hospital clinic, often lacking in adequate facilities to deal with such large numbers. The lowest possible stillbirth rate, however, cannot be obtained by good obstetric care alone, no matter how excellent such care may be. For this, a higher level of health among mothers would be required, spacing of babies at more favourable intervals should be advised, and adequate advice on contraception should be given.

SUMMARY

A brief report on the stillbirths occurring at the teaching units of the Obstetric Department of the University of Cape Town is presented. Similar figures for the Corporation of the City of Cape Town and private maternity institutions in Cape Town are analysed.

A classification of the causes of stillbirth is given, based on clinical findings.

Chronic ill-health accounts for 40·6% of the stillbirths, followed by the hazards of labour in 38·2%, this high loss being in the main due to the large percentage of 'emergency' cases admitted to the teaching hospitals. Unknown causes of stillbirth account for 15·8% of foetal loss (in 71% of these cases the foetus was macerated). There was 2·5% loss due to miscellaneous causes.

Maternity hospital patients can be divided into 3 socio-economic groups:

1. 'Upper class' women delivered in private maternity hospitals, in whom the stillbirth rate was 12·5 per thousand.

2. 'Middle class' patients delivered in teaching hospitals and mainly White, in whom the stillbirth rate was 22·6 per thousand.

3. Patients living in extreme poverty, of non-White race and with a high incidence of chronic ill-health, in whom the

stillbirth rate was 50·5 per thousand. This type in the main was delivered at the obstetric teaching units.

It is suggested that this high stillbirth rate which particularly is associated with the non-White can be remedied in 2 ways, viz. (1) by improving the socio-economic conditions under which these women are living, thereby combating chronic ill-health, and (2) by providing adequate hospital accommodation so as to minimize the adverse effects of 'emergency' admissions.

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ACETAZOLAMIDE (DIAMOX) IN THE TREATMENT OF EPILEPSY

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The enzyme carbonic anhydrase is present in high concentration in the erythrocytes, the renal cortex, and the gastric mucosa,[1] and plays an important part in maintaining the pH of the blood at its proper level by catalyzing the reversible reaction $CO_2 + H_2O \rightleftharpoons H_2CO_3$. In 1940 Mann and Keilin[2] found that sulphanilamide had a powerful inhibiting effect on the production of carbonic anhydrase, and it has been shown by Southworth[3] and Marshall *et al.*[4] that both experimentally and clinically it could produce a marked acidosis and an alkaline urine. A study of other sulphonamides revealed that acetazolamide (Diamox, Lederle) was 50-400 times as active as sulphanilamide as an inhibitor of carbonic anhydrase. Acetazolamide is a white crystalline solid with the empirical formula $C_4H_6N_4O_3S_2$, and chemically is 2 acetylamino-1, 3, 4-thiadeazole-5 sulphonamide. It is soluble in water and remarkably non-toxic even in large doses. In animals, absorption after oral administration is complete and about 60% of the drug is eventually excreted in the urine accompanied by marked diuresis.

In man the production of carbonic anhydrase in the blood is reduced to one third of its normal value by the giving of 8-16 mg./kg. of acetazolamide per day. Yet another effect of the drug is to inhibit the secretion of hydrochloric acid in the stomach.

Three main clinical fields for the use of Diamox thus present themselves from a study of its effects, viz. (*a*) to produce acidosis, (*b*) to produce diuresis, and (*c*) to diminish the gastric acidity.

Research work in all these fields is going on actively, although the work on gastric acidity has apparently not yet gone beyond the laboratory stage. In this paper we are particularly concerned with the production of acidosis, for this was shown many years ago to have a beneficial effect on epilepsy, and the ketogenic diet as a means of treating epilepsy enjoyed a considerable vogue for a time. It has been largely abandoned, not because it was ineffective, but because it was found to be too difficult for an epileptic to adhere to this regime. Considerable success with Diamox has, however, also been achieved as an oral diuretic in conditions such as congestive cardiac failure,[5-10] glaucoma,[11-13] chronic emphysema,[14] and oedema of pregnancy.[15] It has also been used successfully in the treatment of such conditions as hepatic cirrhosis, nephritic oedema, hypertension, and oedema resulting from the administration of cortisone.

Diamox in Epilepsy

A number of clinical trials have been carried out with Diamox in the treatment of epilepsy. Mostly the drug has been given in combination with the patient's basic anticonvulsant treatment, but Riives[16] administered it alone to a group of patients, of whom only one showed a favourable response. The majority had an increased number of seizures

TABLE I. NUMBER OF SEIZURES

No.	*Sex*	*Age*	*Race*	*Diagnosis*	50 *Days before Diamox; on Anti-convulsants only* (1)	50 *Days on Diamox plus Anti-convulsants* (2)	50 *Days on Anti-convulsants only* (3)	50 *Days on Diamox only* (4)
1	M	14	E	Imbecile	9	53	9	48
2	M	18	E	Idiot	2	0	3	X
3	M	10	E	Idiot	25	7	36	56
4	M	14	E	Imbecile	2	0	0	1
5	M	12	E	Imbecile	36	36	31	44
6	M	12	E	Imbecile	44	26	59	Y
7	M	7	E	Imbecile	3	0	0	0
8	M	5	E	Imbecile	5	7	2	7
9	M	6	E	Idiot	4	0	7	3
10	M	6	E	Idiot	5	3	10	X
11	F	5	E	Idiot	1	0	4	1
12	M	27	E	Imbecile	21	14	14	48
13	M	27	E	Feeb.	7	2	5	8
14	M	29	E	Imbecile	5	0	0	Z
15	M	22	E	Imbecile	9	1	68	13
16	M	19	E	Feeb.	14	2	5	X
17	M	18	E	Feeb.	11	9	17	32
18	M	25	E	Imbecile	120	23	31	22
19	M	36	N	Ep. Psychosis	13	4	5	4
20	M	31	N	Ep. Psychosis	6	1	4	4

X = Patient had so many fits that the injection of sodium Gardenal was necessary. Thereafter omitted from clinical trial.
Y = Omitted from final stage of trial owing to his poor physical condition.
Z = Discharged on leave of absence.

when placed on Diamox alone; when combined with phenobarbital or Sodium Dilantin the seizure rate decreased considerably.

Merlis[17] studied the effect of Diamox on 47 patients over a period of 11 months and found that 29 showed a marked improvement.

Ansell and Clarke[18] state that Diamox may be of value as the sole therapeutic agent in the treatment of major epilepsy of moderate severity, in some cases of minor epilepsy, and as adjuvant therapy in mixed idiopathic epilepsy.

Lombroso *et al.*[19] administered Diamox to a group of 127 patients with various forms of epilepsy and found that it gave practically complete control in 34 cases, with a 90-99% reduction of fits in an additional 12 cases. In no case was the condition made worse.

Bergstrom *et al.*[20] showed, in a group of 42 patients with intractable epilepsy, a 90-100% seizure control in 4 cases, and a 50-90% control in another 4 cases. None were made worse by the treatment.

TABLE II. WHITE-CELL COUNT

(Thousands)

No.	*Before Diamox; on Anti-convulsants only*	30 *Days after Diamox plus Anti-convulsants*	*Before the administration of Diamox only*	30 *Days after Diamox only*	30 *Days after conclusion of Clinical Trial*
1	10·9	8·0	7·4	7·5	4·2
2	14·1	10·8	11·5	7·9	10·8
3	8·0	12·0	13·8	9·9	9·4
4	11·9	14·6	16·4	10·2	8·5
5	X	X	11·6	11·1	12·4
6	12·6	7·7	9·6	Y	Y
7	10·3	12·0	13·8	11·9	15·6
8	12·2	12·0	14·8	6·3	15·9
9	10·1	12·4	16·6	9·2	4·0
10	7·9	13·8	11·4	Y	11·4
11	X	X	19·1	14·3	9·2
12	7·8	7·1	11·0	7·1	9·8
13	6·7	9·4	9·6	6·0	12·0
14	6·4	5·4	8·0	Z	Z
15	12·4	9·8	10·1	6·8	7·9
16	5·5	3·8	9·7	5·7	8·5
17	7·4	6·9	7·1	9·9	8·1
18	5·7	5·1	5·6	5·2	5·8
19	6·8	4·1	8·7	4·0	5·2
20	7·8	7·0	6·6	3·6	10·6

X = Unable to obtain blood.
Y = Omitted from final stage of trial owing to poor physical condition.
Z = Discharged on leave of absence.

THE EXPERIMENT

No. of Seizures

It was therefore decided to test the efficacy of Diamox on a number of epileptics in this institution, both when given as an addition to the anti-convulsants already being used and when used by itself. For this purpose 20 epileptic patients of various types were selected, some being mental defectives and others mentally disordered (see Table I). An arbitary period of 50 days was selected for the duration of each section of the trial, as being long enough to give a reliable result. Furthermore, our initial supply of Diamox happened to be sufficient only for this period. The number of fits each patient suffered during each period was carefully recorded, but no distinction was made between different types of fits (grand mal, petit mal, etc.) because no provision for this had been made in the institution records before Diamox was used.

The drug was given in a dose of 12 mg. per kg. body-weight per day, this being the average dose used in previous trials by other investigators. No serious side-effects were noticed; 3 patients showed some temporary fatigue during the first few days of treatment, 1 patient showed short-lasting excitement for several hours after the first 4 doses, and 2 patients developed diarrhoea lasting 2 days at the beginning of treatment. A careful check was kept on temperatures, but no rises were observed.

Blood Picture

Agranulocytosis has been recorded by Pearson *et al.*[21] during the administration of Diamox. Davidson *et al.*[22] found no haemopoietic effects after 3 years' experience with Diamox. Riives[16] likewise failed to discover any blood abnormalities in his cases. All our patients had white-cell counts done before commencement of treatment with Diamox. After 30 days on Diamox plus anti-convulsants this was repeated. Further counts were done immediately before treatment with Diamox alone, 30 days after commencing treatment with Diamox, and 30 days after withdrawal of Diamox. Thus each patient has altogether 5 blood counts during this trial (see Table II).

STATISTICAL NOTE

The most appropriate test for distinguishing the general effect of different treatments would seem to be the Sign Test for Paired Observations.[23] As an example of its use consider the first two columns of Table I. The first pair of observations is 9, 53 which scores 1, since $53>9$. The second pair is 2, 0, which scores 0, since $2>0$. We score each pair in this way, counting 1 if the second observation is greater, and 0 if the first observation is greater. Let the total number of pairs be n, and the final score r. If two numbers are equal we shall score $\frac{1}{2}$, unless both are zero, when it seems better to ignore that pair (e.g. for the first two columns $r=2\frac{1}{2}$, $n=20$).

We set up the null hypothesis that the two treatments are equivalent. In this case r should be distributed according to the Binomial Distribution $(\frac{1}{2}+\frac{1}{2})^n$ .... There are tables which give the probability of r or less numbers of the second column being greater than the corresponding numbers of the first column.[24] If this probability is small, we regard our null hypothesis as disproved, and this gives an indication that one treatment is superior to the other. The tables only give integral values of r, so to be on the safe side in Table III Row (*a*) we take $r=3$, whence, from tables, $P=\cdot0013=0\cdot13\%$. This is a very small value and so we have confidence in saying that the addition of Diamox has a beneficial effect.

TABLE III

Columns	*No. of Cases n*	*Score r*	*P*	*Better Treatment*
(*a*) 1, 2 ..	20	3	0·13%	Col. 2 **
(*b*) 2, 3 ..	17	13	2·45%	Col. 2 *
(*c*) 3, 4 ..	17	11	16·60%	Col. 3 ?
(*d*) 1, 3 ..	20	9	41·19%	Col. 3 ?
(*e*) 1, 4 ..	19	12	17·96%	Col. 1 ?
(*f*) 2, 4 ..	17	14	0·64%	Col. 2 **

Note: In Table I, Column 4, X is regarded as a large number, and hence e.g. (10, X) scores 1. Y and Z have been omitted.

No. of Seizures

We have compared columns in Table I in all possible ways, and indicate the results (Table III) by stating the apparent 'better' column, with a question mark if the result is not significant (this means that no conclusion ought to be drawn, and further experimentation is necessary), with one, two, or three asterisks if the result is significant at the 5%, 1%, or 0·1% levels respectively. The more asterisks the greater confidence one has in the results.

Conclusions (Tables I and III)

From Rows (*a*) and (*b*) there seems little room for doubt that the addition of Diamox to the usual treatment is an improvement.

From Rows (*b*) and (*d*) it appears that the effect of Diamox lingers on to a slight extent after the treatment has stopped, although the effect is so small that it is not significant.

From Row (*f*) it seems that Diamox alone is definitely inferior to Diamox plus anti-convulsants, and Rows (*c*) and (*e*) suggest that Diamox alone may even be inferior to anti-convulsants alone, though again the results are not significant.

White-cell Count

To estimate variations in the white-cell count we compare adjacent columns of Table II (Table IV).

TABLE IV

Columns	*No. of Cases n*	*Score r*	*P*	*Lower Count*
(*a*) 1, 2 ..	18	6	11·9%	Col. 2 ?
(*b*) 2, 3 ..	18	15	3·8%	Col. 2 *
(*c*) 3, 4 ..	17	2	1·2%	Col. 4 *
(*d*) 4, 5 ..	17	11	16·6%	Col. 4 ?

Conclusions (Tables II and IV)

Rows (*b*) and (*c*) show a significantly lower white-cell count when Diamox is being given. The same trend appears from Rows (*a*) and (*d*) but the difference is not significant.

DISCUSSION

A number of interesting facts can be deduced from a study of the figures in Table I. In the 50-day period before Diamox was begun, and while the 20 patients taking part in the trial were on their usual anti-convulsants, they had a total of 342 fits between them, or an average of 17·1 fits each (column 1). When Diamox was added to their former drugs (column

2), the total fell to 188 for a corresponding period, or an average of 9·4 fits each. Furthermore, 6 of the patients had no fits at all while on anti-convulsants plus Diamox, while a further 6 had 4 fits or fewer. The total in this group was raised considerably by the fact that case 1 had an attack of status epilepticus during this period and this brought his fits alone up to 53. Altogether 17 out of the 20 patients had fewer fits after Diamox was added than they had on their basic anti-convulsant treatment. As has been shown above the improvement produced was statistically of considerable significance, and the probability of this result being due to chance was only 13 in 10,000—a very small figure indeed.

When Diamox was stopped (column 3), the position reverted very much to what it had been before it was added, although the total fits amounted to 310, i.e. 32 less than before the Diamox had been given, with an average of 15·5 fits per patient. Furthermore 3 patients had no fits at all during this period, and there is a faint suggestion, perhaps borne out by the statistical investigation, that there may have been a beneficial 'carry-over' effect from the Diamox after it had been stopped.

On Diamox alone (column 4), the total number of fits among 17 patients (3 patients having dropped out for various reasons) was 291 or an average of 17·1 fits per patient, a figure identical with that obtained on basic anti-convulsants alone (*v.s.*). Only 1 patient had no fits during this period while a further 5 patients had 4 or fewer.

One must therefore conclude from this trial that Diamox added to the basic anti-convulsant is a valuable drug in reducing the frequency of fits, but that by itself it has no advantages over anti-convulsants.

The average white-cell count of the patients taking part in this trial (see Table II) was 9·1 thousand on anti-convulsants, 9·0 thousand when Diamox was added, 10·6 thousand when they returned to their basic anti-convulsant treatment, 8·0 thousand on Diamox alone, and 9·4 thousand when Diamox was stopped. All count were done at the same time of the day and at the same time in relation to meals. There was thus a reduction each time the patients took Diamox, and an increase when it was discontinued. The reduction when Diamox was added to the former treatment was statistically significant but never fell low enough to give rise to a fear of agranulocytosis. Even more significant was the fall in the count when Diamox was given alone, and during this period the lowest average count occurred, although in no case did it fall to dangerous levels. It was noticed that the 2 lowest counts of 4·0 and 3·6 thousand (cases 19 and 20) were in the only 2 non-Europeans (Natives) taking part in this trial. One cannot of course draw any conclusions from only 2 cases, which may have been chance results, but it is possible that Diamox may reduce the white-cell count more severely in Natives than in Europeans.

SUMMARY

Acetazolamide (Diamox) was administered to 20 epileptic patients in combination with their usual anti-convulsants, and by itself, for periods of 50 days each. A very significant reduction in the number of fits occurred when Diamox was added to the anti-convulsants, but not when it was given by itself.

A reduction in the white-cell count occurred when Diamox was given; this was most marked in non-Europeans.

There appears to be an indication for the administration of Diamox in the treatment of epilepsy.

We should like to express our gratitude to Alex Lipworth Ltd. (Distributors for Lederle Laboratories), Johannesburg, for a generous supply of Diamox for the purpose of this clinical trial.

We are grateful to Dr. I. R. Vermooten, Commissioner for Mental Hygiene, for permission to publish this paper.

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ASSOCIATION NEWS : VERENIGINGSNUUS

EAST RAND BRANCH

At the 7th annual general meeting of the East Rand Branch of the Medical Association of South Africa, which was held at the Boksburg-Benoni Hospital, Boksburg, on 24 January 1957, Dr. S. Copans took the chair and 31 members were present.

The Secretarial Report and Financial Report were presented by Dr. W. M. Bezuidenhout (Hon. Secretary and Treasurer). The membership of the Branch had risen in 1956 from 194 to 213. The attendance at Branch meetings had been unsatisfactory although outstanding clinical lectures and discussions had been held; of the 10 meetings 2 had been cancelled; the average attendance was 16. The Branch Council had held 12 meetings, and tribute was paid to the work of the sub-committee (Drs. Gould, Karstaedt, Goldblatt and Goldberg) in investigating the operations of benefit and medical aid societies on the East Rand. The social functions during the year included a party and dance at the Springs Country Club (the total proceeds of which are to be donated to the Benevolent Fund), and golf competitions.

The Hon. Secretary also reported that members of the Eastern

Transvaal Division of the Southern Transvaal Branch were in favour of incorporation of that Division in the East Rand Branch, and it was decided to press for this change in boundaries.

The Branch Council for 1957 was elected as follows: *President*, Dr. E. Meltzer; *Vice-president*, Dr. A. Karstaedt; *Hon. Secretary/Treasurer*, Dr. W. M. Bezuidenhout; *other members*, Drs. I. Goldblatt, J. Noll, A. Gould, L. E. Haine and E. King. (*Federal Council members*, Drs. J. Q. Ochse, E. W. Turton and M. Segal.)

Medical Auxiliaries. It was agreed in principle that medical auxiliaries should be registered by Medical Council and that the register should be a statutory one.

Association Medals. It was decided to draw up a memorandum asking Federal Council to amend the method of awarding medals.

New Members. To avoid delay it was decided that the Branch Constitution should be altered so that the Branch Council should have the power to approve the appointment of new members.

Grants. It was decided to make a grant of £35 to the library and £5 to the Marriage Guidance Council.

Induction of President. In inducting Dr. E. Meltzer as President the retiring President (Dr. Copans) spoke of him as a stalwart of the Branch and said he had long deserved this honour. Dr. Copans then delivered his valedictory address (which is published at p. 567 of this issue of the *Journal*), after which Dr. Meltzer presented to him a gold medal from the Branch.

A vote of thanks was passed to the Superintendent of the Boksburg-Benoni Hospital for the courtesy and hospitality accorded to the Branch.

ASSOCIATION NEWS : VERENIGINGSNUUS

HEALTH, HEALING AND HAPPINESS *

S. COPANS, M.B., CH.B.

President, East Rand Branch

The choice of a subject for a valedictory address is a matter of some difficulty, particularly for a general practitioner, a President of a Branch of the Association which consists almost entirely of general practitioners, either in private practice or employed in Provincial and Mine Hospitals. In common with my fellow members, I have little time to spare for research or more than cursory references to recognized authorities and works. I am sure that most of my fellow colleagues these days find it no easy matter to keep up with the few publications that are essential to a high standard of knowledge of recent advances. In choosing the title of 'Health, Healing and Happiness', I can only present some thoughts and impressions that have evolved in my mind since I have been in practice.

S. Copans

In our profession, with its many divisions into specialities, the general practitioner could easily be looked upon as specialist for the whole family, who is expected to have some knowledge of all the ailments that may affect its members, and who is in the best position to maintain and restore the happiness of the people he is serving.

HAPPINESS THE CONCERN OF THE DOCTOR

Jean Jacques Rousseau defined happiness as a state obtained 'by a good bank account, a good cook and a good digestion', but we should have a more accurate and less satirical definition than this. On reference to the various dictionaries at my disposal, I found great difficulty in finding a suitable definition, at least to suit my mind, until I looked up the meaning of the words 'health' and 'heal'. 'Health' is defined as 'a state of bodily or organic soundness, freedom from bodily or mental disease or decay'. 'Heal' is defined as follows: 'To make whole or to restore to health, to cure.' Happiness, I would say then, means a little more than possessing a good bank balance, good food and digestion; it also means being blessed with physical and mental well-being. The possession of a sound body and contented state of mind must contribute a great deal to a happy state for the individual. As doctors we can only assist in the attainment of the latter two. We exert very little influence as individuals, or as an organized profession, on the economic and social factors that affect mankind and, so often, are responsible for his ill-health and his misery. Even if a man enjoyed that Utopia of favourable conditions in which he was assured of agreeable employment and adequate remuneration, of ample time for recreation, vacation and culture, there is no guarantee that he would make good use of them. How often as a profession do we observe this misuse by people who are 'well off and short of nothing'. As doctors we shall always have to treat ailments caused by man's own abuse and misuse, albeit very often unwittingly. However, much illness, misery and distress are avoided today by preventive and prophylactic measures, and the family doctor also participates in this, e.g. by advice in the campaigns of prophylactic vaccinations and inoculations; but it is the general practitioner's role in relation to the happiness of the family and the patient that is the theme of this address.

As a student I cannot remember any of my teachers stressing the fact that when we qualified we had a particular role to play in practice—that of contributing to the happiness of our patients. The text-books we studied made no mention of this; no more, I believe, do the text-books of today. A description of a complaint is divided into the usual headings of 'definition', 'aetiology', 'morbid anatomy', 'clinical picture', 'treatment', etc. The heading 'prognosis' however, touches indirectly on this question of happiness of the patient and those concerned with him. In the giving of the prognosis, or rather the manner in which it is approached, much can be contributed to the immediate happiness of all concerned, or at least to the cushioning of the despondency and dejectedness that bad news might precipitate.

If the doctor is the friend of the family, as he should be, he should know how to put the position, how to raise justifiable hope, how to break bad news gently to the patient and to the relatives, or even to conceal the truth, at least temporarily. As, fortunately, not all afflictions are fatal or crippling, we still have to bear in mind that in the handling and treatment of even minor ailments we have a role to play in this respect. Sometimes, of course, the patient is only happy when he or she has some complaint, real or imaginary, and here is encountered a different problem.

ILLUSTRATIVE CASES

Mrs. A.B., who had a long medical, surgical and gynaecological history, was eventually referred to a urologist for a renal calculus, for which a left nephrectomy was done. A short time after this she presented herself with haematuria and a story of pain in the other kidney. We were told that she had had blood transfusions when in Johannesburg while under further specialist care but

* Valedictory Address delivered at the annual general meeting of the East Rand Branch.

when we checked with the consultant, he denied this but said he was told we had given blood transfusions. We were at a loss to account for the bleeding and were extremely puzzled until one of the sisters recognized the patient as one who had been previously in another hospital, many years ago, and was then found to have in her possession a knitting needle which accounted for the bleeding of a suspected threatened abortion. Some time later, the patient presented herself to us again with a typical history of diabetes mellitus and urine loaded with sugar. It soon became apparent from the normal blood sugars and severe Benedict's reaction that, even in hospital, sugar was being added to the specimens. She admitted to this deception and was sent home. Her next admission was for burns to both legs, said to have been caused by the spilling of battery acid, but in view of her previous history and the nature of the lesions, we were certain that these were self-inflicted and, on being taxed with this, she admitted to this new deception. Some of her other complaints may have been suspect, but these three were outstanding and proved. It was thought that the patient was a drug addict, but this was later discounted. The outstanding feature, however, was her very happy state while in hospital as opposed to the state of misery she always presented at home or at the consulting room.

After the last episode, this patient agreed to an abreaction treatment under mild narcosis and then divulged that she became very unhappy at home when her husband's drinking became excessive. She indulged in all these subterfuges to gain admission into hospital to get away from him and thus obtain temporary happiness. Since then we do not think she has had self-induced or self-inflicted complaints, although she has had much further hospital treatment. In spite of our efforts and explanations, I do not think she ever acquired real happiness until she obtained a divorce.

This is an example of a patient who sought the hospital for happiness. Some seek admission to hospital not to escape from their surroundings, but to obtain the false happiness and escape induced by drugs. For the majority of people, however, the word 'hospital' certainly does not spell happiness and this is a thought which we often overlook when advising admission to a nursing institution to heal them and restore their health.

As an example, take the child admitted to a hospital. There is no single person to give him such care as he is used to at home and my experience has been that children, particularly babies, take a long time to get used to their new surroundings. At the same time, the anxious and worrying parents have a very harrowing time on being parted from their sick child, made worse by the thought that things must be serious if the child is in hospital. Similarly, elderly people are usually thrown into great consternation at the mention of hospital. There are, however, a number of old people, particularly those living alone in boarding houses or in flats, who are only too anxious to be admitted to a hospital, even when they have a minor complaint not warranting this step. When these are admitted, the nursing staff are resentful and unhappy and, I should say, with some justification.

Except when absolutely necessary, our aim should be to keep patients out of hospital and treat them at home. The ideal, of course, would be to prevent sickness, so that there would not be any need for treatment either in hospital or at home. But even treatment at home demands care in order to create as much happiness as possible for all concerned, irrespective of the gravity of the illness. Similarly in our consulting room we should be on our guard not to cause needless dismay or despondency. Colleagues will have observed how some patients, tranquil at the commencement of a consultation, become alarmed because one has perhaps listened too long to a particular area in the chest or heart or palpated the abdomen for some time, or even at the routine taking of the blood pressure. One wonders, in this connection, how many people have been made unnecessarily miserable because they have been informed that they suffer from 'high blood pressure'. I still treat an old lady of 79 who was found to have a blood pressure of 220/120 in 1937. My outlook in those days was not what it is today but, fortunately, she was a sensible person and was not upset by my information. In all this period, she has had very little treatment until recently, when she was given small doses of a rauwolfia product, which only upset her. She regained her former comparative well-being as soon as treatment was stopped, and is now quite happy again. I would give you two further examples of this—those of two patients aged 54 and 70 who became extremely unhappy and depressed while taking the same drug. One, in fact, had to have electric shock treatment before she recovered. Fortunately, both are now well and of a happy frame of mind and are not taking any drugs at all and I am sure are in no danger from what I would label as their 'compensated hyperpiesis'.

MINERS' PHTHISIS

Of a different category are those patients who seek further advice, because, on routine examination, they have been informed that they have some condition which is looked upon as a deviation from normal health. I have in mind here particularly the miner who hardly consults us for years, and then only for minor complaints, and then comes to us in haste, in great distress and much disturbed, because he has been informed that he is suffering from some stage of silicosis. Whereas, before that date, they felt well, had no complaints and were quite happy, they have now become anxious and distressed and many of them complain now of various symptoms. Some it is possible to persuade to disregard the diagnosis labelled onto them and they regain their previous happy outlook on life and retain a justifiable optimism. Others, unfortunately, go downhill in their despondency, especially if they are no longer able to carry on with their previous occupation, with its higher income. Yet I must say, although I know I am generalizing, in my experience, except when tuberculosis supervenes, these people seldom become gravely ill, and the outlook even for the silico-tuberculotic is now much better with the advent of new drugs. In my early days of practice, I had many silicotic patients who were employed on what was then a non-scheduled mine; those who are dead died either from tuberculosis or some other unrelated condition. Many, however, including healed tuberculotics, are still alive, and my impression is that explaining the position helped many to throw off despondency and alarm and adjust themselves to an acceptable existence. I could quote actual case histories, but time does not permit.

While one realizes that there is no alternative to the law and that the beneficiaries have to be advised accordingly when they are found to be suffering from silicosis (or pneumoconiosis, as it is now known), much could be done by both lay and medical officials of the responsible bodies to avoid needless anxiety and distress in the handling of these people in their examinations and their notifications. You, no doubt, have had the experience of being consulted by a patient in great distress, firmly convinced that there must be something seriously wrong with him, because the doctor at the Bureau spent longer than usual in examining him, or he was sent back for further X-rays, or he was referred to another doctor, etc., etc. These people, even after a careful examination by us in which no deviation from health can be found, take a lot of convincing that they are well and have no cause to be unhappy about themselves.

SCHOOL MEDICAL INSPECTION

A further example of this type of occurrence is the notification parents get from the school medical inspectorate. You are all aware of the very curt official form, with its bare diagnosis and occasional sparse comment. Recently some acquaintances of mine were caused a grave anxiety until their daughter was examined by the paediatrician that had seen the child since birth, who was able to assure them that although, there was a murmur to be heard in the heart, this was not a lesion, as was suggested in the official note. You all, no doubt, have also been visited by anxious parents with similar notes about other conditions, especially enlarged tonsils, and have also had to allay unnecessary distress.

Whilst acknowledging that these periodic examinations at the school undoubtedly uncover defects and conditions that are the better for being discovered and treated early, I think that a little more tact and discretion is called for in the handling of the examinations and in their notification. Nevertheless, in those cases where a serious illness is prevented, the school doctors are contributing to the essential happiness of the family.

DISCRETION IN TREATMENT

Whilst it is true that, if we prevent or heal, we preserve or restore health and confer a better chance to achieve happiness, nevertheless we should gravely consider whether it is wise to advise or insist on a treatment, when to many the treatment is worse

than the condition; and often experience and statistics make it doubtful whether a cure can be achieved.

Mr. G. J. was found to have a carcinoma of the stomach for which a total gastrectomy was performed. He survived for a year after his initial operation, during which period he lived a miserable existence, sedated by opiates and wishing only for the return of his stomach so that he could enjoy his food. False hopes had been raised in his family, who suffered an unhappy period watching his pain and misery and then queried the wisdom of the major operation and of the stormy convalescence and the year of suffering.

In contrast to this case, which entailed so much investigation, treatment, expense and anxiety and then an unsuccessful outcome, I should like briefly to relate the history of another patient without I hope, being thought cynical. This patient, a woman seen some years ago, complained of vague indefinite symptoms of lethargy, but really consulted me for periods of oligomenorrhoea. No gynaecological cause could be found and the patient was given dried thyroid extract, of which she is now having 5 gr. every morning. She was a mild case of myxoedema, and has become a changed, happy person. Her happiness is reflected in her home. In this case much has been achieved in what might have become a very serious irreversible condition by very little trouble. The lesson, if any, to be learnt here, is that it is not always necessary to subject our patients to elaborate and expensive procedures to restore them to health.

It is, of course, not suggested that we abandon thoroughness where this is needed, but to plead that we must be certain of the need before subjecting patients to intensive and often costly investigations. Some of these are not without risk. One need only mention the fatalities that have occurred with oesophagoscopy and liver biopsies. Sometimes the risks may be latent, as exemplified by the suggestion put forward recently that there is a higher incidence of leukaemia in children who were subjected even to small doses of X-ray in antenatal investigation.

Zeal in treatment may sometimes be the cause of some unhappy permanent affliction. One has only to think of the poor unfortunates who have retrolental fibroplasia as the result of the excessive administration of oxygen to the premature infant, or perhaps of the intractable skin conditions precipitated by gold therapy, or even the loss of life, fortunately not so frequent now, caused by arsenical injections. We are now continuously being reminded of sudden deaths that occur with penicillin. Perhaps not so serious, but nevertheless most uncomfortable, are the side-reactions of oral antibiotics, and one has only to have suffered the discomforts of a pruritus to appreciate the consideration one should exercise before deciding to give a patient antibiotics or sulpha drugs for a minor complaint. In the urge and inclination to do something, because we think it is demanded of us, let us try and remember—not always easy when busy—that many of the treatments we order are not without risk and that, instead of creating health, we may precipitate unhappiness.

Doubtless you all remember the misery and unhappiness undergone by the infant who had Pink disease, and its mother. Here at least we can take some credit for discovering the cause and stopping the treatment of infants with teething powders containing calomel.

THE DOCTOR'S OWN HAPPINESS

Before concluding, I think it would not be out of place to refer to the happiness of the doctor himself in this triangle, because that must surely be of some importance if the best is to be achieved for those entrusted to his care. To me it is obvious that if the doctor, whether he be specialist or general practitioner, in full-time employment or private practice—is healthy, happy and contented with his work, he will be able to render good service to his patient. He is, of course, subject to the same conditions that afflict his patients. He is perhaps more prone to the degenerative diseases. He should, with his knowledge, be able to avoid and prevent or detect early deviations from health, but it is notorious that he pays very little attention to himself. If he does, he usually goes to the other extreme and is almost hypochondrial. But assuming good health, then his happiness must largely depend on his work, the manner in which he is able to carry it out, and the remuneration it brings him. This should be adequate and equitable and should allow him not only a decent standard of living, but also to make provision for illness and retirement. It is not pleaded that he should seek wealth out of his work, but that he is entitled to a decent living, and to attain this should not work excessive hours; that he should have some order in his calling and his life; that he should have some time to spare for further study, during his working days and in postgraduate courses; and that there should be sufficient time for recreation, vacations and the pursuit of culture.

His patients, or rather the public generally, can do much to contribute to his happiness. He, in turn, could then accomplish more for them. Nevertheless, whether or no he receives such consideration, it behoves him in his work, no matter how trivial or serious the condition he is called upon to treat, to bear in mind that there is much he can do for the happiness of those he is meeting in his calling. Although he is constantly being exhorted by the State, industry and commerce to restore the health of the people speedily so that they can return to their occupations and contribute to the general welfare of the community, he still has a primary duty to his patient, and if he bears all this in mind and accomplishes it successfully, he will not only see contentment and happiness in his patients, but also participate in these feelings himself.

VERLOSKUNDIGE EN GINEKOLOGIESE KONGRES

OPENINGSTOESPRAAK *

SY EDELE DIE ADMINISTRATEUR, MNR. P. J. OLIVIER

Ek kan my nouliks 'n vertakking van die mediese wetenskap voorstel wat meer belangrik en meer onontbeerlik is vir die opbou van 'n gesonde en lewenskragtige nasie as die Vereniging van Verloskundiges en Ginekoloë. 'n Gesonde moeder en 'n gesonde baba verseker die voortplanting van die gemeenskap en van die volk. Die uitoefening van u beroep en spesialiteit is van onmeetbare waarde en betekenis vir ons land. Dit verheug my om te kan getuig dat die dae van wanpraktyke, onhigiëniese toestande, onkunde en bobbejaanvangers haas verby is. Daarvoor is die volk en die nageslag dank aan u professie verskuldig. Alte veel kosbare lewens is onnodiglik in die ou dae verloor voordat u gespesialiseerde dienste algemeen beskikbaar was.

As leek is ek wel deeglik bewus van die feit dat ek my bevind in die geselskap van gespesialiseerde professionele persone aan wie ek beswaarlik beroepsvoorligting of mediese raad kan gee. Dit is 'n geval van skoenmaker hou jou by jou lees. U moet my derhalwe verskoon as ek my liewer nie te diep op die terrein van die mediese wetenskap begewe nie. Ongetwyfeld sal ek 'n foutiewe diagnose maak en die verkeerde behandeling voorskrywe.

*Kaapstad, 16 April 1957.

VOORUITGANG IN VERLOSKUNDE EN GINEKOLOGIE

Ek vertrou egter dat u my sal vergun om in die verbygaan 'n paar woorde te sê in verband met ginekologie en verloskunde. My Administrasie, as beherende liggaam van provinsiale hospitale en kraaminrigtings in Kaapland, is wel deeglik gemoeid met die welsyn van u Vereniging. Ons is wedersyds afhanklik van mekaar en dit is my innige wens dat ons verhoudinge en samewerking in die toekoms net so aangenaam en vrugbaar sal wees soos dit nog altyd in die verlede was.

Dit is opvallend en verblydend om te sien watter geweldige vooruitgang daar gedurende die afgelope halfeeu was op die gebied van die mediese wetenskap in die algemeen en van die spesialiteit ginekologie en verloskunde in besonder. 'n Mens dink onwillekeurig in dankbare herinnering aan die wetenskaplikes wat sulke kostelike bydraes gelewer het op die gebied van mediese

navorsing. Vandag besit u nie alleen 'n wyer kennis van u beroep nie, maar u beskik ook oor verbeterde metodes van diagnose en behandeling wat, gepaard met strenger voorsorgmaatreëls, nuwe geneesmiddels en meer doeltreffende apparaat en instrumente, baie van die vrees, lyding en menseverlies uitskakel. Die hedendaagse neiging is in die rigting van spesialisasie en alhoewel 'n mens soms effens heimwee voel oor die krimpende geledere van die algemene praktisyn wat in die reël die vertroulike familievriend is, is dit nietemin 'n voorwaartse stap en in belang van die pasiënt en van die mensdom.

TEKORTKOMINGE IN SUID-AFRIKA

Ten spyte van die feit dat daar in ons tyd wonderlike vordering in ons land gemaak is ten opsigte van die voorsiening van gesondheidsdienste, besef ek maar te goed dat daar nog baie leemtes bestaan wat 'n stremmende uitwerking op ons pogings en ideale het. Dit sal geduld, tyd, inspanning, samewerking en veral middele en werkkragte kos om naby die gewenste toestand te kom, soos dit byvoorbeeld in die Skandinawiese lande bestaan. Ons moenie uit die oog verloor dat ons land nog betreklik jonk is en dat ons met talryke vraagstukke te kampe het—onder andere, die klimaat, uitgestrektheid en geografiese geaardheid van ons land, lang afstande, vervoerprobleme, rasse-aangeleenthede, die tekort aan opgeleide verpleegsters en mediese hulppersoneel en die noodsaaklikheid om binne ons geldelike vermoë en belastingsbronne te bly. Ongelukkig bestaan daar nog 'n sekere mate van oorvleueling en soms verwarring tussen die Unie-regering, die Provinsiale Administrasies en plaaslike owerhede wat die verskaffing van gesondheidsdienste betref. Maar laat ons ons troos dat Rome nie in een dag gebou is nie. Die posisie is kortliks, soos u weet, dat die Unie-regering verantwoordelik is vir rehabilitasiedienste en vir die hospitalisasie van persone wat aan geestesverstorings ly, die Provinsies vir genesingsdienste wat distriksverpleging, buitepasiëntdienste en die hospitalisasie van nie-aansteeklike gevalle behels, en plaaslike owerhede vir voorkomende of verhoedende gesondheidsdienste en vir die hospitalisasie van aansteeklike gevalle.

Alhoewel uiters nuttige dienste in die kraamwêreld alreeds aan verwagtende moeders verskaf word deur klinieke en distriksverpleegsters, wat almal geregistreerde vroedvroue is, voel ek nogtans dat die voor- en na-geboorteversorging van moeders veel te wense oorlaat. Daar is ook ander verskynsels wat die prentjie nie te rooskleurig laat voorkom nie en waaraan vermoedelik min of niks gedoen kan word nie. Dit ly geen twyfel dat daar 'n groot persentasie verwagtende moeders van alle rasse in ons land moet wees wat hoegenaamd geen mediese of verplegingshulp versoek of ontvang nie. Daar rus in elk geval geen verpligting op hulle om deskundige hulp in te roep nie en daar bestaan ook geen wetlike voorsiening waarkragtens hulle daartoe gedwing kan word nie. Ek wil u net 'n voorbeeld noem. Volgens die 1955-sensus was daar in die Bellville-Magistraatsdistrik 5,500 geboortes van alle rasse vir daardie jaar. Van hierdie getal is 4,000 bevallings deur geneeshere en/of geregistreerde vroedvroue waargeneem. Dit beteken derhalwe dat 1,500 gevalle (dit is 27%) òf geen mediese of verplegingshulp ontvang het nie, òf deur ongekwalifiseerde persone behandel is in daardie distrik gedurende 1955.

ROL VAN PROVINSIALE ADMINISTRASIE

Soos ek reeds gesê het, my Administrasie is verantwoordelik vir hospitalisasie van nie-aansteeklike gevalle en derhalwe stel hy om klaarblyklike redes baie belang in die wel en wee van die mediese beroep. Sonder geneeshere kan ons maar net so wel die deure van ons hospitale sluit. Soos u natuurlik weet is dit 'n wetlike vereiste dat geneeshere minstens 'n jaar na graduasie hul internskap by 'n hospitaal moet dien voordat hulle geregistreer kan word en mag praktiseer. Die Administrasie voorsien ook geriewe by sy hospitale vir die opleiding van mediese studente van die Universiteite Kaapstad en Stellenbosch en vir nagraadse studie. Afgesien van mediese ere-personeel, is daar 'n taamlike aantal gesalarieerde geneeshere in die diens van die Administrasie. So byvoorbeeld is daar op die diensstate van die Departemente Ginekologie en Verloskunde by Groote Schuur-Hospitaal 8 voltydse en 14 deeltydse geneeshere en by Karl Bremer-Hospitaal 3 voltydse geneeshere en 3 interns. Ek meen stellig dat die Provinsiale Administrasie nie hoef te bloos vir die fasiliteite wat hy tot u beskikking stel nie en vir sy aandeel in u opleiding en praktyk.

HOSPITALIZATION

I now proceed to a subject where I am on safer grounds. As you know my Administration is responsible for the erection and maintenance of provincial hospitals and maternity homes. Private hospitals and nursing institutions may not legally take in patients until they have been registered by me, and then the Director of Hospital Services and his authorized officers have the right to inspect any private hospital. Where I now refer to hospitals, I have in mind provincial and private institutions subsidized by the Administration.

Maternity Beds

In regard to normal confinements you are no doubt aware that unless there are complications my Administration regards a confinement as a normal physiological event and not as an illness or disease which makes it imperative for the patient to be admitted to a hospital. For this reason the Administration has made provision for such cases in its hospitals and will continue to make such provision when planning new hospitals. When plans are drawn up for a new hospital or when it is intended to erect additions to existing hospitals and it is considered necessary to provide for gynaecological and obstetrical cases, it is the aim of my Administration to keep abreast of the most recent developments of the medical world and to meet the demands of our gynaecologists and obstetricians. Full-time medical practitioners, architects and engineers of the Administration have on several occasions, and at a considerable cost to the Administration, been sent overseas to make a study of the newest trends and developments in hospitalization. Many normal confinement cases are admitted to our hospitals, especially in country districts where there are no maternity homes. In support of this statement, I should like to quote some illuminating figures. On 31 December 1956 the rated number of beds in provincial hospitals in the Cape Province was 7,823, of which 677 were maternity beds—a percentage of 8·6 maternity beds. During 1955 a total of 171,909 in-patients were admitted to provincial hospitals in the Cape Province, of whom 25,099 were confinement cases—i.e. a percentage of 14·6 confinement cases. In subsidized private hospitals in the Cape Province the rated number of beds on 31 December 1956 was 2,368, of which 214 were maternity beds—i.e. 9%. During 1955 a total of 38,938 patients were admitted to subsidized private hospitals in the Cape Province of whom 8,828 were confinement cases—i.e. 22·6%. I think you will agree that the number of confinement cases admitted during 1955, namely 14·6% at provincial hospitals and 22·6% at subsidized private hospitals, reflects a healthy state of affairs. Unfortunately, I am unable to furnish any statistics in connection with fee-paying private hospitals and maternity homes.

In passing, I should like to mention that recently 48 maternity beds were made available at the Karl Bremer Hospital, Bellville—30 for Europeans and 18 for non-Europeans. In addition, considerable progress has been made with the erection of a maternity block with about 50 beds for Europeans and 50 beds for non-Europeans at the Groote Schuur Hospital, Cape Town. Not only will these beds supply a need, but they will also come in useful in the training of midwives and medical students.

NURSES AND MIDWIVES

A while ago I referred to the shortage of trained nurses. Notwithstanding improved salary scales and more attractive conditions of service, there remains a shortage in the approved establishment of all branches of the provincial nursing service, with the exception of the unqualified nursing assistants. The conditions, especially at some of our country hospitals, give cause for alarm. Perhaps you will be interested to know how many nurses there were in the provincial service at the end of last year. The figures are as follows:

	European	*Non-European*
Registered nurses*	1,174	223
Registered midwives	18	17
District nurses†	71	53
Student nurses	1,763	786
Student midwives	155	119
Qualified nursing assistants ..	69	20
Unqualified nursing assistants..	1,004	185

*Medical and surgical, with a large proportion also registered midwives.

†All registered midwives and some with the double qualification.

I regard the considerable number of student nurses as a very gratifying indication that our efforts to make the nursing profession more attractive were not in vain. We must naturally be prepared to accept that some of these students will drop out in the course of their studies or will resign and that some will join private hospitals and institutions and nursing agencies upon completion of their studies. There is nevertheless a marked improvement. It is my policy to make increased provision and to create more facilities for the training especially of non-European nurses in order to enable them to look after their own people and thereby relieve the pressure on the European nurses.

My Administration encourages its nurses to improve their qualifications by means of postgraduate study and for this purpose study leave on full or part salary is often granted. Upon completion of their postgraduate studies special grants are paid in certain cases to those nurses who make use of their additional qualifications in our hospital service. So, for example, general nurses are encouraged to obtain the additional qualifications for which they will receive recognition, viz. diplomas in nursing and certificates in midwifery, mothercraft, nursing of children's diseases, theatre technique, practical ward administration, and infectious diseases.

DISTRICT NURSING AND OUT-PATIENT SERVICES

I cannot refrain from mentioning a development in which, I am sure, you will be interested. Last year the Union Government transferred the administrative and financial responsibility for district nursing and out-patient services to the Provinces. As you may probably know, the Provinces carried out these services on behalf of and at the financial responsibility of the Department of Public Health. Now the Province will have to foot the bill itself. How this will work out in practice remains to be seen but, personally, I feel optimistic about the scheme. In future it will not be necessary for the Provinces to go hat in hand to the Department of Public Health every time they find it necessary to introduce urgently required new services or to expand existing services.

In conclusion, after welcoming members of the Congress to the mother city of Cape Town and wishing them a pleasant and fruitful time, Mr. Olivier declared the Congress open.

NAUDÉ VERSUS WHITTLE

IN THE SUPREME COURT OF SOUTH AFRICA (EASTERN DISTRICTS LOCAL DIVISION)

JUDGMENT DELIVERED 29 APRIL 1957

Sampson, A. J.: Plaintiff, a young doctor in practice at Franklin, who is also District Surgeon for the Franklin and Zwartberg areas of the Mount Currie district, claimed damages in the sum of £5,000 from Defendant, a farmer in the Zwartberg area on account of a defamatory statement alleged to have been made by Defendant at the annual meeting of the Zwartberg Farmers' Association.

In Paragraph 6 of the Declaration the defamatory statement is set out as follows:

'When Mr. George Cullen of the New Amalfi area committed suicide on the night of 13 October 1955, the District Surgeon, Franklin (meaning and referring to the Plaintiff) was telephoned at 10.30 p.m. and refused to appear at that hour, although the circumstances were explained.'

Paragraph 7 of the Declaration then added eight innuendos, of which I shall only set out three:

'7. (*b*) That the purpose of the said telephone conversation with the Plaintiff was to request him to proceed to the scene of the tragedy and there to give such professional services as he could and was obliged to do in his capacity as a medical practitioner, and as the District Surgeon for the area concerned.'

'7. (*d*) That the Plaintiff refused to respond to the said request to render such professional services on the ground merely that it was late at night and that his refusal therefore was without good and sufficient reason or excuse'; and

'7. (*e*) That therefore Plaintiff was guilty of a serious dereliction of his professional duties as a medical practitioner and as a District Surgeon.'

Paragraph 8 alleged that Plaintiff has suffered in his personal and professional capacity as a medical practitioner damages in the sum of £5,000.

Defendant denied speaking or publishing the words alleged and therefore denied the innuendos alleged in the Declaration; and alternatively he denied that the words, if he did publish them meant or were understood to mean what was alleged in the innuendos. Further Defendant pleaded in the alternative that, if he did publish them as alleged, and if they did bear the meaning assigned in the innuendos, he had published them on a privileged occasion, without malice and in the *bona fide* belief that they were true.

PRIVILEGE PLEA

The grounds for this claim of privilege were pleaded as follows:

'(*a*) Defendant has at all relevant times been a farmer in the Zwartberg area of Mount Currie District, and a member of the Zwartberg Farmers' Association.

(*b*) The said association is a voluntary association of farmers in the said area, whose objects *inter alia*, are
  (i) to serve as a mouthpiece of local farmers and to act on their behalf;
  (ii) to deal with, further and protect the interests of the farming community;
  (iii) to do all things which in the opinion of the members are in the interest of the farming comminity.

(*c*) In terms of the constitution of the said Association all matters affecting the said Association and its interests shall be dealt with at the General Meeting of the said Association.

(*d*) On 27 October 1955, the General Meeting of the Association was held at Zwartberg and was attended by Defendant and the persons mentioned in paragraph 5 of the Declaration all of whom were members of the Association.

(*e*) The professional conduct of the District Surgeon for the said area was a matter affecting the interests of the farming community of the said area, in particular the members of the said Association.

(*f*) The said words published by Defendant related to the professional conduct of the District Surgeon for the said area.

(*g*) The said words were published by the Defendant at the said General Meeting without malice and in the *bona fide* belief that they were true.

(*h*) It was the duty of Defendant to publish the said words to members present at the said General Meeting, and the said members had a lawful interest in having the said words published to them'.

To this an alternative ground of privilege was pleaded in that 'the said words were published by Defendant in furtherance of his interest as a member of the said Association, and the other members present at the said meeting had a lawful interest in having the said words published to them'.

Defendant denied the allegation that Plaintiff had suffered damages to his good name and reputation in his personal and professional capacity in the sum of £5,000; and finally denied that his refusal to pay any part of the said sum was wrongful or unlawful.

THE INNUENDOS

Mr. O'Hagen, Q.C. for Defendant did not seriously contest the justice of the innuendos and Defendant himself did not in evidence make any bones about admitting that he had intended

to convey to the meeting what he was alleged to have said, although he was positive that he did not say expressly that 'the District Surgeon had refused to come although the circumstances had been explained'. Defendant told the Court that he reported to the annual meeting that he and Stubbs had endeavoured to 'phone to the Police at Banchory and Zwartberg and at Cederville without success, as the Exchange had failed to plug these stations through for the night; that finally about 10.30 p.m. they got through to Kokstad; and that he then listened at the earphone while Stubbs spoke to the man on duty asking him, after they were told not to touch the body, to get someone to come out to the body and to notify the District Surgeon at Franklin; that this man then undertook to do so; that they waited until 1 a.m. when they rang Kokstad again and were told by the man on duty that he was still attempting to get some policeman to come out; and that when Stubbs enquired about the District Surgeon, the reply was that the District Surgeon will not come out till tomorrow morning. Defendant's evidence was that this was what he said to the meeting about the District Surgeon and nothing else. This account of his words does not quite tally with the terms of his report as set out in the minutes of the meeting; for the minute reads that 'they were informed that Kokstad was unable to contact any policeman to send out and that the medical officer at Franklin had refused to come'. I gained a strong impression of Defendant's probity, so much so that I am satisfied that if he had had any doubt of the actual words he had used he would have said so. For this reason I take the view that the terms of the report in the minute reflect the effect of the actual words as this effect was voiced in the discussion which follows. I consider that the construction thus put upon Defendant's words were justified. Mr. Mortlock also said that Defendant had said that the Police reply from Kokstad was that the District Surgeon refused to come out. I am satisfied that Mortlock, a striking figure of brusque honesty, is mistaken in some of his evidence, e.g. about the presence of Stubbs at the meeting; and he probably is mistaken here too. Dorning, who was elected President of the Association at this meeting for the ensuing year, says that he got the impression that Defendant was concerned about the failure to get the District Surgeon, and that he, Defendant, considered that they should know in the interests of the community how far his obligations went. The minute shows that the meeting decided, after discussion, that a letter be written to the Magistrate about the District Surgeon and that a communication be sent to the District Controller of Posts and Telegraphs about the telephone difficulty.

COMMON CAUSE

It was common cause that Plaintiff was never notified of Cullen's death during the night on which it occurred, or of the request that he should come out to the body. On the morning of the 14th the Police gave him information that a post-mortem would be required which he mistakenly understood referred to a Coloured person. Plaintiff first became aware of what had happened at the General Meeting of the Association when he received the Magistrate's letter dated 31 October (Exh 'D'). This letter conveyed to Plaintiff the precise terms of the Defendant's alleged statement to the Association as it appears in the Declaration in the form of an extract from a letter addressed to the Magistrate by the Secretary of the Association; and it called for Plaintiff's comments. It upset Plaintiff very much, so much indeed that he was unable to complete a letter in reply. In this state of mind he telephoned to several people and eventually spoke to Defendant. He told Defendant who he was and that he had received a letter from the Magistrate and understood he (Defendant) had brought the matter up. Defendant admitted that he had done so and added that he was not a young man but a responsible member of Society and could not make such a charge without sufficient ground. Plaintiff then said he would not leave the matter there, and to this Defendant made the cryptic reply that he did not see why those chaps should get away with it. Plaintiff did not ask what he meant being apparently too distressed to take the words in; but he told Defendant that there was not a word of truth in the statement, that he was at home all night and was never contacted. Later Plaintiff got Mr. Strachan on the 'phone and told him about the trouble and that he could prove that he was never notified; that he did not want to go to law; and he asked Mr. Strachan for his advice what to do to clear his name, and he complained that no one had been to him first before the letter to the Magistrate was written.

On 5 November 1955, the District Commandant, South African Police, wrote to the Secretary of the Association in reply to the latter's letter of enquiry that the District Surgeon at Franklin had not refused to attend to the late Mr. Cullen; and that the Charge Office at Kokstad had 'phoned to the Police at Franklin and requested them to raise either Banchory or Zwartberg Police on the telephone and to inform the District Surgeon of the incident; that the telephone line was out of order and neither of these stations could be raised; and that after he had been personally informed of the circumstances and had satisfied himself that life was extinct he had ordered that the District Surgeon should not be contacted until further instructions from himself. It is very regrettable that this last piece of information was not conveyed to Mr. Stubbs and Defendant. The letter concluded with the statement that the District Surgeon at Franklin was never notified of the death of the late Mr. Cullen by any member of the Police, and with an expression of regret if the misunderstanding had caused any inconvenience.

It is clear that the statement about Plaintiff made to the General Meeting of the Association on 27 October 1955, was a highly misleading one which conveyed implications altogether untrue, implications which Defendant has said he intended to convey. These implications were of a defamatory nature and there has been no attempt on the part of the Defendant to show that they were not defamatory. The only serious defence to the resultant claim for damages is that of qualified privilege; in other words it is contended that the occasion of Defendant's statement was a privileged occasion and that Defendant made the statement without malice and in the *bona fide* belief that what he said was true.

Counsel were only able to refer to one case in which the issue on this defence arose in respect of a statement at a meeting of a farmers' association by a member of the association, viz. *Philpott* v. *Whittal, Elston & Crosby and Co.* (1907 E.D.C. 193 at 204) and in that case KOTZE, J. P. found it unnecessary to decide the question whether the occasion was privileged as the case went off on the ground that express malice had been established. I am thus left with the difficulty described by SCRUTTON, L. J. in *Watt* v. *Longdon* (1936, 1 K.B. 130 at 144) and in a number of English cases there cited. The *desiderata* for the existence of a privileged occasion are set out as follows by WATERMEYER, A. J. A. (as he then was) in *De Waal* v. *Ziervogel* (1938 A.D. 112 at 120 *et seq.*): 'where a person publishing the defamatory matter is under a legal, moral or social duty to do so, or has a legitimate interest in so doing and the person to whom it is published has a similar duty or interest to receive it, then the occasion of the publication would be privileged.' I shall not presume to add any words of my own to this definition, and shall only say that I understand the word *interest* to mean what is popularly meant thereby, as suggested by Gatley, 4th edition, p. 202.

THE FACTS EXAMINED

I should now examine the facts in order to ascertain whether any such duty or interest existed for the Defendant and the Association or its members as is required to support the plea. *Mr. Cloete*, Q.C. for Plaintiff contended that Defendant's interest and the interests of the farmers, members of the Association, was no other than the interest of the public to see that a public official did his duty, and on this basis he contended that Defendant could only have claimed qualified privilege in respect of a report to some person who had authority to enquire into the official's conduct and to correct it if necessary. For this he relied upon *Gatley 4th Edition at p. 226 and the case quoted in note 16 to that page*, and he submitted that members of the public, whether they were farmers or not, could not by banding together in an Association such as the Zwartberg Farmers' Association give to a meeting of that Association the legitimate interest required. In other words, as I understand his argument, publication to such an association would be too wide, for he says one would not be entitled to go to one member of the public after another with the defamatory statement and claim that each such publication was covered by qualified privilege. The action of the Defendant in the present case can I think be distinguished from a purposeless series of publications to the individual members of the Association. *Mr. Cloete* contended that there was no special or particular interest which farmers in the Zwartberg area could have in the conduct of the District Surgeon. I think, however, that their comparative isolation on the farms and the presence there of the

farm servants and their families exposes them to the incidence of emergency at night to a special extent, in the form of the onset of infectious or contagious disease, accidents or the urgent need of medical assistance owing to criminal assaults. The very fatality which gave rise to Defendant's report may well have been one which called urgently for the attendance of the District Surgeon, if the body of Mr. Cullen had been found with life still present; and as *Mr. Cloete* has argued the real gravity of the defamation cannot be evaded by a plea or consideration that the report was of a complete or effective suicide, because that gravity resides in the implication that his conduct in refusing to come out late at night is the result of a settled intention so to conduct himself. This was the matter of concern to Defendant of which Mr. Dorning speaks. The District Surgeon at Franklin was the only medical practitioner within reasonable call, the next nearest being Cedarville. The special interest of the farmer members of the Association may be seen also reflected in the minutes—the Association was apparently largely responsible for the appointment of a District Surgeon; at any rate, it is clear that the Association agitated strongly in the matter. *Mr. Cloete* says that the Association could only act as the *mouthpiece* of the farmers. This very function of a farmers' Association has become widely recognised as a salutary feature of State life, or community life, and there can be no doubt that such an Association, the product of the modern genius for organisation, provides the farmer with a weighty and effective voice. Defendant did what the constitution of the Association recognised; he brought the matter before the General Meeting so that a complaint about it to the authorities might rest on the responsibility of the Association. It is true that this interest of the Association which I have illustrated above, is not the every day interest of a farmers' association, but the ambit of legitimate interest should not in my opinion be restricted on that account. I have come to the conclusion that I should hold that the occasion of Defendant's statement to the General Meeting was a privileged occasion, for there can be no doubt that Defendant himself shared the interest of the Zwartberg farmers. I do not think that the fact that the District Surgeon's contract requires that he should act on instruction of the proper authority in the matters I have mentioned above makes any real difference to the interest of the farmers of the Zwartberg area in his attitude towards his duties. I have no doubt whatever that Defendant was not actuated by any sinister, improper motive or malice. Plaintiff has failed to establish anything of the sort. Defendant's statement to the Court that he would not apologise obviously rested upon his feeling that he had done no more than his duty to his fellow farmers, and cannot be relied upon as evidence of malice.

CLAIM DISMISSED

For the foregoing reasons I consider that Defendant's statement was made on a privileged occasion without malice and in the *bona fide* belief that it was true.

If, however, I am wrong in finding that the occasion of its publication was privileged, I would after consideration of all the circumstances—the absence of apology in public, the failure of Plaintiff to respond to the offer contained in the letter of 15th December, 1955, the fact that Mr. Dorning says that when he stated to the meeting of the Association in January that the report was false, practically everyone was aware of the fact, and the consideration that the publication did not have a very wide ambit of interest and was probably quickly corrected so far as the Franklin and Zwartberg area was concerned—allow in damages the sum of £800.

The claim is dismissed with costs. The Defendant is declared a necessary witness.

[Information has been received that an appeal against this judgment has been noted. — Editor.]

NOTIFICATION OF MALARIA

In connection with the compulsory notification of Malaria (all forms) the Secretary for Health has issued the following request to medical practitioners:

(*a*) Medical practitioners should indicate whether the malaria was contracted locally or outside the boundaries of the Union, and the probable place of infection should be stated.

(*b*) Medical practitioners should also indicate which malaria parasite (*Plasmodium falciparum, P. vivax, P. malariae* or *P. ovale*) caused the disease.

(*c*) Medical practitioners should place at the disposal of the Government laboratory the positive blood smears or duplicate thereof (at least one thick smear).

OBSTETRICAL AND GYNAECOLOGICAL CONGRESS

E. M. SANDLER, HON. SECRETARY, CAPE TOWN BRANCH OF SOUTH AFRICAN SOCIETY OF OBSTETRICIANS AND GYNAECOLOGISTS

A combined congress of the South African Society of Obstetricians and Gynaecologists and the South African Regional Council of the Royal College of Obstetricians and Gynaecologists was held in Cape Town on 16-18 April 1957.

The Congress was opened by the Hon. the Administrator of the Cape Province, Mr. P. J. Olivier, on Tuesday 16 April. His address is published elsewhere in the *Journal*. Dr. J. C. Coetzee occupied the Chair throughout the Congress.

The following papers were read:

1. *Manual removal of the placenta without general anaesthesia* by Dr. H. Claassens, University of Cape Town.
2. *Persistent occipito-posterior position* by Dr. H. V. F. Jordaan, University of Cape Town.
3. *Invitation paper: The problem of intersex* by Dr. R. Hoffenberg, University of Cape Town.
4. *Invitation paper: Vaginal hysterectomy; its ididcations and some points of technique* by Dr. John Stallworthy, Director, Area Department of Obstetrics and Gynaecology, United Oxford Hospitals (UK) and Visiting Professor, University of Cape Town.
5. *The etiology of genital prolapse* by Professor F. Geldenhuys, University of Pretoria.
6. *Constriction ring dystocia* by Professor James T. Louw, University of Cape Town.
7. *Coarctation of the aorta in pregnancy* by Dr. F. Daubenton, University of the Witwatersrand.
8. *Trace elements in the so-called toxaemias of pregnancy* by Dr. D. P. de Villiers, University of Cape Town.
9. *Eclampsia* by Dr. H. Mukheiber, University of Cape Town.
10. *Invitation paper: The physiological changes wrought by operations* by Professor J. H. Louw, University of Cape Town.
11. *The value of basal body temperature recordings in gynaecology and obstetrics* by Dr. F. Benjamin, Port Elizabeth.
12. *Posterior colpotomy puncture* by Dr. P. Bremer, University of Pretoria.
13. *Peritoneoscopy* by Dr. P. F. Venter, University of Pretoria.
14. *Is the art of obstetrics being lost?* by Dr. J. O. E. Apthorp, University of Cape Town.

The social activities included a garden party given by the Hon. the Administrator of the Cape Province and Mrs. P. J. Olivier at Leeuwenhof, their official residence in the Gardens, and a cocktail party given by His Worship the Mayor and Mrs. P. J. Wolmarans in the City Hall, Cape Town.

The Congress Dinner was held in the Students' Union at the University of Cape Town. Among the guests were the Hon. the Administrator of the Cape Province and Mrs. P. J. Olivier, His Worship the Mayor of Cape Town and Mrs. P. J. Wolmarans, the Acting Principal of the University of Cape Town and Mrs. James, and Dr. and Mrs. John Stallworthy.

A Ladies' Committee under the able direction of Mrs. James Louw entertained the wives of visiting members and kindly provided the refreshments for members attending the Congress.

PASSING EVENTS : IN DIE VERBYGAAN

South African Medical Congress, Durban. All members intending to take part in the Bowls Trophy Tournament which will be held during the Medical Congress on Thursday afternoon, 19 September 1957 are requested as soon as possible to notify the Convener of the Bowls Tournament, Mr. B. W. Franklin Bishop, F.R.C.S., at 112 Medical Centre, Field Street, Durban. Telephone 2-4049.

* * *

British Bursary for Postgraduate Clinical Study in the United Kingdom. Applications for this Bursary, which has been established by grants from B.P.D. (South Africa) (Pty.) Ltd., British Drug Houses (South Africa) (Pty.) Ltd., Distillers Company (Biochemicals) Ltd., Evans Medical Supplies Ltd., I.C.I. South Africa (Pharmaceuticals) Ltd. and the Crookes Laboratories Ltd., are invited from general practitioners who have been in active practice in South Africa for at least 10 years. The Bursary, which has a total value of £600, is intended for postgraduate clinical study, and not for research. It is available for not less than a 3-month period in the United Kingdom. No payments will be disbursed to the successful applicant until he has been accepted for the period of postgraduate study at an institution approved of by the Selection Committee. The successful candidate must undertake to return to South Africa for a period of at least 1 year after the termination of the award.

The candidate must submit a brief statement of his proposed course of study and indicate the institution at which he proposes to undertake it. The Selection Committee will consist of an entirely independent board of medical practitioners. Applications must be made on the prescribed form, which is obtainable from Dr. H. A. Shapiro, Honorary Chairman, Selection Committee, British Bursary for Postgraduate Clinical Study, P.O. Box 1010, Johannesburg. The closing date for applications is 31 July 1957.

* * *

Dr. B. Crowhurst Archer, of Durban, has been elected a Corresponding Fellow of the American Psychiatric Association. This award was made 'in recognition of meritorious contributions to Psychiatry'.

Dr. M. Cecil Michelow, M.B., B.Ch., D.Obst. (R.C.O.G.), M.R.C.O.G., who has spent 4 years in the United Kingdom completing his postgraduate training, and who until recently held the post of Assistant Obstetrician and Gynaecologist, Baragwanath Hospital, has joined Drs. S. Joel Cohen and A. M. Porter in Specialist Obstetric and Gynaecological practice at 208 Medical Centre, Jeppe Street, Johannesburg, and 1 Admirals Court, Rosebank.

Telephone: rooms 23-7124, 42-1248; residence 41-4105.

Dr. M. Cecil Michelow, M.B., B.Ch., D.Obst. (R.C.O.G.), M.R.C.O.G., wie vir vier jaar in Groot-Brittanje gewerk het vir verdere opleiding in Ginekologie en Verloskunde, en tot onlangs Assistent-Ginekoloog en Verloskundige by Baragwanath-Hospitaal, praktiseer tans met Drs. S. Joel Cohen en A. M. Porter as Spesialiteit in Ginekologie en Verloskunde by 208 Mediese Sentrum, Jeppestraat, en 1 Admirals Court, Rosebank.

Telefoon: kamers 23-7124, 42-1248; woning 41-4105.

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Cape Western Branch. A Dance will be held at the King David Country Club (near D. F. Malan Airport) on Saturday, 15 June 1957. Tickets are available from the Branch Office, 35 Wale Street, Cape Town (telephone 3-1574).

* * *

South African Dermatological Society. A meeting of the Cape Town Sub-group of this Society will be held at 8 p.m. on Thursday, 20 June 1957, in the small A-Floor lecture theatre at Groote Schuur Hospital, Cape Town. Dr. Sidney Stein will show coloured slides of his cases. All members of the Medical Association will be welcome.

* * *

Dr. M. M. Suzman, of Johannesburg, left for London on 26 May to be present at the Comitia of the Royal College of Physicians, London, at which he will be admitted to the Fellowship, and to attend the Harvey Tercentenary Congress at the Royal College of Surgeons, England, on 2-7 June. Dr. Suzman expects to be away for about a month.

REVIEWS OF BOOKS : BOEKRESENSIES

HYPNOSIS IN ASTHMA

Hypnosis in Asthma. By A. Philip Magonet, B.Sc., M.D., C.M. Pp. 95. 7s. 6d. London: William Heinemann Medical Books Ltd. 1955.

Contents: 1. Anatomy. 2. Etiology. 3. Therapy. 4. Hypnotherapy—Children. 5. Hypnotherapy—Adults. 6. Conclusion. Lecron and Bordeaux System. Glossary. Bibliography. Index.

To me this seems to be a highly biassed little book. It is written from the standpoint that the sole cause of asthma lies in a disturbed psyche. Various figures are quoted to 'prove' that the ordinary forms of treatment are useless, but the larger part of the book is devoted to a citation of cases of asthma which have been cured by hypnosis. No serious worker in this field would deny the great importance of the psychological both in the diagnosis and in the treatment of asthmatic patients, but to suggest that psychotherapy, applied through the medium of hypnosis, has superseded all other forms of treatment, seems to be an extravagant claim.

N.V.S.

CORRESPONDENCE : BRIEWERUBRIEK

LICENSING OF X-RAY MACHINES

To the Editor: A great deal of publicity has been given recently in both the medical and lay press to the dangers of radio-activity from natural and artificial sources to the well-being of the present and future generations of mankind. A great deal of work has already been done in estimating radio-activity in man's natural environment.[1] These factors cannot be altered, but the alarm expressed in the numerous articles which have appeared concerns the increase in the artificial radiations made by man himself since the beginning of this century.

Amongst the causes of this increase is the fall-out from the atomic bomb explosions, which must depend on the number of bombs the USA, Russia and Britain, and possibly other countries, will in future explode, the creation and use of artificial radio-active substances, and the ever-increasing use of essential diagnostic and therapeutic X-rays.

It appears that little can be done by Governments under present international political conditions to reduce the radiation dangers from atomic bombs. As long as the present political unrest exists it is unlikely that any one country which has atomic bombs will agree not to test them.

The use and creation of radio-active isotopes is already very stringently controlled. The only control which is lacking is over X-ray machines for diagnosis or therapy, and their use by medical practitioners and even by unregistered practitioners and footwear specialists.

Most of the dangers associated with the use of either diagnostic or therapeutic X-ray machines are well known and methods have been devised and are continually being improved to reduce these dangers.

One no longer sees on the hands of radiologists X-ray burns finally leading to carcinoma of the hands. It is unfortunately a

type of carcinoma which metastasizes much more readily than the usual epithelioma of the skin. Most radiation burns that are seen nowadays are on the hands of doctors and others untrained in the use of X-ray machines.

LEUKAEMIA

The dangers to the haemopoietic system have long been known and again radiologists have taken precautions for many years to protect themselves and their staffs. These dangers have been greatly exaggerated and an erroneous impression has been given of the incidence of this effect of radiation. Leukaemia is said to occur more frequently amongst radiologists than the rest of the medical profession, and amongst the rest of the medical profession to a higher degree than amongst the public. It has been suggested that the increase in leukaemia amongst medical practitioners other than radiologists is due to its occurrence in medical men who use X-ray machines in private practice or in small hospitals which do not employ radiologists.

The incidence of leukaemia must vary from country to country because of the different working conditions in X-ray departments. The modern X-ray department in South Africa is quite differently equipped and is placed in entirely different surroundings from the old departments in the twenties, say, in Great Britain and elsewhere. Many of them were placed in the basement, with poor ventilation and poor lighting facilities. In South Africa the risk of leukaemia amongst radiologists must be very small, because one does not know of any radiologist who has died of leukaemia at any time and to my personal knowledge certainly not since 1930. I have only heard of one radiologist who has developed leukaemia, and he is of the younger generation and has not done anything like as much radiological work as some of the older radiologists still in practice.

The risk to radiographers is negligible under present conditions, whether in hospital or in private practice. I have had to investigate a number of reports during the war years that radiographers and nurses near the X-ray departments or working in X-ray departments had become anaemic. In no single instance could the slight anaemia be definitely traced to any work within the X-ray department or to proximity to the X-ray department. The suggestion was made in the Johannesburg *Sunday Times* of 12 May 1957 that radiographers in private practice were not so well protected as the radiographers in hospital practice. The *Sunday Times* is not the only place where this suggestion has been made. Radiographers have apparently been advised not to work for private radiologists because of this danger. This suggestion, of course, is quite unjustified because many of the radiologists in private practice in Johannesburg have had more experience than any radiologist at the Johannesburg Hospital and many others have actually worked at the Johannesburg General Hospital.

In the practice of radiotherapy, measures have been taken to protect the radiologists and other personnel. Reactions which appear in patients in the course of treatment are deliberately caused, and the patient very rarely gets any radiation reaction which is not intended by the radiologist. Again the risk of causing leukaemia in the patient must be extremely small, even in the treatment of patients with spondylo-arthritis ankylopoeitica, who seem particularly susceptible to the effects of radiation. It is noted that Dr. Durbach,[2] in his extensive but uncritical summary of the literature on the subject, does not indicate how many cases of leukaemia have been found amongst the patients treated at the Johannesburg General Hospital. I must assume as Dr. Durbach has taken so much trouble and devoted so much time to compiling his review of this subject, that had there been any cases of leukaemia at the Johannesburg General Hospital he would have drawn attention to them. Presumably, then, there have been no such cases. Moreover, hundreds of thousands of cases have been treated with radiotherapy for various conditions throughout the world and, if the radiation was an important factor in causing leukaemia amongst these patients, it would have become very obvious. One has not heard of leukaemia occurring amongst either radiotherapy or diagnostic radiographers.

EFFECTS OF PUBLICITY

The publicity given in the press to these risks has done a considerable amount of harm in various directions. Patients in whom genetic effects could not take place because of their age have been worried about having a barium-meal examination. Extraordinary memoranda have appeared suggesting that a single X-ray examination of the chest once a year is dangerous. Patients who have been treated for conditions such as syringomyelia with the beam applied to the cervical spine have enquired whether they should give up the idea of getting married and whether they are likely to develop leukaemia.

These reports also have an unfortunate effect on employees in the X-ray departments and private practice.

Professor Sievert,[3] in an article on Tolerance Levels in Swedish Radiation Protection Work states: 'If sufficiently strong warnings are given it is difficult to get employees. If insufficient warnings are given people will work with inadequate caution.' The difficulty of getting radiographers will be increased if this fear of the effects of radiation is exaggerated and publicized.

CONTROL OF DANGERS

Although it is relatively easy to protect personnel and patients against the usually well-known dangers of radiation, it is not so easy to protect them against the genetic effects because not enough is known of the subject. G. Pontecorvo[4] states: 'It is clear that at present we know nothing of the dynamics of cell population which links an irradiated cell of today with a genetically determined death, disability or change in later generations.'

Whatever dangers there are due to radiation, it is obviously essential to reduce them to a minimum. If Governments cannot control dangers from atomic bombs, they can control the dangers due to ignorance of the effects of radiation. The best way to do that is to license X-ray machines so that any one wishing to buy or use a machine must give proof that he has had sufficient training. It seems an extraordinary state of affairs that to get a minute quantity of radio-active substance, which may only be a beta emitter, one has to get a special permit, and yet any medical practitioner or layman may buy an X-ray machine without regard to his knowledge of the subject. Only certain radiotherapists are granted permits to obtain radio-active substances; even radiologists who have been in practice for many years have to apply for a permit, and it is not always granted.

About 25 years ago at a meeting of the Southern Transvaal Branch of the Medical Association I urged that X-ray machines should be licensed, and I have repeated this a number of times; but nothing has been done. In Sweden since 1926 there has been unofficial supervision of X-ray therapy and since 1941 government supervision of radiation protection in all forms of radiological work; if anybody wants to buy an X-ray machine, he has to get permission from Professor Sievert of the National Institute of Physics. The layman would not get permission at all. It is not a matter of professional knowledge but of technical knowledge concerning protection. I do not know any other country which has adopted this system. In England, since the introduction of the National Health Service, very little private work is being done by radiologists, and none at all by those who are not radiologists. In the USA physicians and surgeons who are not radiologists still use X-ray machines.

The best way to control the amount of radiation which is being added to the natural radiation is not to curb the activity of the radiologist by continually urging him to take fewer films and do less screening. The radiologist is conscious of all these dangers and the danger should not be made an excuse for skimped work. An X-ray examination for possible cancer of the stomach can have no genetic effect on a woman of 50, and if several more films will demonstrate whether the patient has a carcinoma or not, obviously they should be taken. This applies to the examination of any other system or to the treatment of any lesion. The amount of radiation can be cut down to a considerable extent by excluding all those who have no training or experience in the use of X-ray machines. There is no reason, for instance, why shoe shops should have X-ray machines. The possible genetic danger to a child standing on one of these machines to have his shoes fitted is probably much greater than having his chest screened to exclude some pathological condition. The examination of the feet for the fitting of shoes should be banned forthwith; it is dangerous and only an advertising stunt.

All those wishing to acquire X-ray apparatus (excluding registered radiologists) should, after a fixed date, have to apply for a licence before they are allowed to buy the machines, whether they are for screening purposes only, or for radiographic work,

or for X-ray therapy. The date suggested is 1959 so as not to interfere with the established rights of medical practitioners who already have machines; the machines, however, should be under periodical inspection.

The idea of licensing machines is so obvious that one wonders that only in Sweden has it been brought into force. What is the value of articles published in the medical and lay press pointing out (and often exaggerating) the dangers, if the simplest and most obvious steps to reduce these dangers are not taken?

If it is dangerous to have a chest X-rayed once a year, or a lumbar spine X-rayed, or a cervical spine or shoulder treated with small doses of X-ray then all machines should be scrapped. In fact there are no dangers in such procedures; but that is no reason for not taking the greatest precautions when working with X-rays or radium or isotopes. Yet it is inconceivable that even the most enthusiastic supporter of protective measures or the most timid worker in an X-ray department would wish to return to the pre-Roentgen days. Radiology has been of inestimable value to mankind, and has done relatively very little injury. One might as well advocate the abolition of antibiotics because a few people have developed reactions to them.

As the use of radiation cannot and should not be abolished, all medically qualified practitioners (other than registered radiologists) who wish to use ionizing radiation for diagnosis or therapy, should be expected to show that their training and knowledge makes them aware of the dangers. There is no justification whatever for the use of X-ray apparatus by lay practitioners such as chiropracters or by foot-wear specialists.

M. Weinbren

Johannesburg
23 May 1957

1. Spiers, F. W. (1956): Brit. J. Radiol., **29**, 409.
2. Durbach, D. (1957): S. Afr. Med. J., **31**, 187.
3. Sievert, R. *in* Haddow, A. ed. (1952): *Biological hazards of atomic energy*, p. 186. Oxford: Clarendon Press.
4. Pontecorvo, G. *in* Haddow, A., *op. cit.*, p. 83.

FIFTY YEARS AGO

Eheu fugaces, labuntur anni
Autres temps, autres moeurs
O tempora, O mores!

To the Editor: To hark back to the happy days of their youth and to contrast them with those of the contemporary generation is a pastime common to most sexagenarians, septua- and octogenarians. The occupation is a harmless one, satisfying to the moralizer, faintly humorous to the more tolerant youngsters who come under inspection for their manners and modes, but it achieves nothing, except an '*eheu fugaces*' in the breast of the 'old 'uns' who may have survived the changes of five reigns, and the violence of two or three wars.

In common with the layman the aging doctor laments the changed mode, the new content of life, and its accelerated pace; all of which add up to the loss of so much that was stately, dignified and peaceful. Gone are some of the little courtesies which prevailed, in public at least, between members of the profession, whose experience and scientific attainments clothed them as with a mantle of dignity which set them apart from the layman. Gone too, in large measure, is the respect for the doctor, and the acknowledgment of his leadership by the 'man in the street'.

Undoubtedly there has been a loss of dignity, of 'face', perhaps due to our wranglings over State and contract practice. The hard-bargaining business man, and the trade-union leader have confronted us with 'solid facts' nor could they ever realize the altruistic or missionary spirit in which almost every doctor did his *pro deo* work, in his own way, in his own time. The control from above, from every side in fact, with restriction and regimentation, is a far cry from the freedom and the joy of work which the ancients among us can recall.

Regard for a moment the city practitioner of the first decade of this century. He was a general practitioner with proclivities in one or more directions within his craft. The only specialists then were those devoted to eyes, ears and throats and, though private rivalries and keen competition existed, the integrity and dignity of the profession was a matter of great unanimity. Those were the days of the frock coat and tall silk hat, which received their quietus with the advent of the motor car. Every city doctor had his 'trap' driven by a coachman in some sort of livery, or at least a cockaded hat, and a brave sight they made as they trotted along Adderley Street or the suburban highways.

The newly arrived young practitioner was duly impressed, nor did he fail to make his bow to seniority by a personal call, which generally established good, often intimate, relations. Contrast this picture with the present method of announcing the advent of a new colleague, usually a 'specialist', by a printed card, stereotyped in style and wording, through the post. The value of a personal introduction is missed, the personality of the newcomer is submerged by his list of degrees and recently-held hospital posts. None can deny that the loss of that intimate contact which alone 'places' the new colleague is a real one. The kindly encouragement of the older colleague is missed by this modern mode, and with it the establishment of personal relations which may be of the utmost value to the young 'un.

This loss of touch at the outset leads to a slackness in the strict observance of medical etiquette when a consultation is called for. How frequently one finds the family practitioner late for the appointment, and the senior may even be asked by telephone to 'carry on' with the examination in the absence of the junior, who was 'detained by urgent work' elsewhere. The rank discourtesy of such conduct seldom seems to occur to the youngster, even when he completely disowns liability for the consultant's fee by asking him to 'send his account direct'.

The sins of neglected correspondence must also lie heavily upon the conscience of the average young practitioner in this our country. It is a form of discourtesy becoming increasingly common nowadays, and it is decidedly worse in South Africa than in other countries. It may be due to the schooling, or to the upbringing, but this pen-shyness is certainly no credit to our manners. I recall vividly the sadness with which an aging London consultant once enquired about a South African practitioner whom he had coached through all his examinations, and had come to love as a younger brother, but who never replied to his letters enquiring about his health and welfare.

But enough of this grousing; I have strained the Editor's hospitality too greatly, and can only say once again—*Eheu!*

Senex

16 May 1957

PROFESSIONAL PROVIDENT SOCIETY

To the Editor: I am one of those who acted on one of the investigating committees of the Dental Association of South Africa when it mooted the idea of establishing a Professional Provident Society. Largely as a gesture in support of the project I took 8 shares and I am one of the 'survivors' of 1941, 17 in number, mentioned in the Society's most recent actuarial report.

At the moment I own 32 shares, which cost me £69 12s. 0d. per annum. I have just received my apportionment account for 1956. It reveals that during the year I was credited with £18 16s. 5d. in interest and £49 0s. 9d. as my share of 'profit', making a total of £67 17s. 2d. for the year.

Thus, during 1956—and I see no reason why the same thing should not hold in 1957, if indeed not more so—I was covered for sickness, etc., to the tune of £20 0s. 0d. per week (32 times 12s. 6d.) This at a cost of £1 14s. 10d. per annum or 1s. 4d. a week. £20 per week for 1s. 4d. per week! Where else could I obtain such terms? In addition, my policy can never be 'called in', and at age 60, 61, 62 or 63, at *my* choice, I shall collect quite a nice little nest-egg of some hundreds of pounds.

I am at an age, 58 plus, when I normally must expect a certain amount of illness, but I am not haunted by the fear that I 'dare not' get ill.

My great regret is that I did not take the maximum number of shares as the available number increased from time to time (at present 50).

I have written this personal note so as to influence junior colleagues to waste no time in joining this great mutual benefit run *by* the profession *for* the profession.

R. Hofmeyr

17 Church Square
Cape Town
23 May 1957